Heart Sounds, Cardiac Pulsations, and Coronary Disease

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by

William Dock, M. D.
Professor of Medicine, State University of New York, College of Medicine at New York City

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Preface

An invitation to give a lecture, and especially to give the Porter Lectures in succession to a distinguished line of physicians and scientists, may well lead to the question as to whether the generous opinion of one's friends, the combined thoughts and labors of one's associates, or one's own contribution to medicine has led to such an honor. There is no doubt in my mind that, in the case of the 1955 Porter lecturer, the first two factors counted heavily. However, in a world where the affection of friends is as important to most of us as is prestige based on good luck or hard work, it is comforting to realize that we have kind friends on the faculty of the University of Kansas and that the efforts of other old friends have contributed to whatever reputation we have achieved

In developing my ideas on the topics discussed in these lectures, I have been greatly stimulated by many other workers, and have been directly aided by Doctors J. K. Lewis, David Rytand, and Herbert Hultgren, of the Stanford University School of Medicine, San Francisco, and by Doctors Joseph Brady, Felix Taubman, Anthony Caccese, Julio Ortiz-Marquez, Alvin Schrager, Harold March, and Francis Grandell, who have been Fellows in the laboratory supported by grants from the National Institutes of Health, U.S.P.H., at Kings County Hospital Doctors Leonard Brandt, Harold Lyons, John J. Kelly, Jr, and Herman Ruskin have provided invaluable data from our patients, and have also supplied information obtained by reading and by contact with other workers in these fields Ancel Keys, Louis Katz, Forrest Kendall, and my colleague, Howard Eder, have tried in vain to

correct my heresies on the subject of cholesterol deposition, and Isaac Starr, Ben Baker, Victor McKusick, Frank Davis, and William Scarborough have offered sound advice about ballistocardiography. Robert Elliott provided me with his neat Ueda-principle accelerometer, M. B. Rappaport with his light, low-frequency bed, and electromagnetic accelerometer, and Samuel H. Boyer assembled the gadget used for producing heart sounds under water. To all of these, and to the friends who have so often heard me talk about these topics—Samuel A. Levine, William Resnik, William Castle, Tinsley Harrison, and the late Harry Mandlebaum—I am deeply grateful.

In the thirty-seven years since, as a sophomore medical student, I first attempted to contribute to the solution of a medical problem, many golden opportunities have been presented to me. Failure to see clearly and act energetically explains why all of the really big fish got away, to be netted by others with quicker wits, greater initiative, or more stern tenacity. What little I have accomplished is certainly due to genes inherited from the McLemores and McMornes, who for over a century had constantly been ready to move over the hills into undeveloped lands, and from three generations of Dock physicians and engineers, who delighted in nature, in the use of delicate instruments and fine tools, and in modifying their professional habits in order to do their work more effectively. Thus conditioned not to accept the current dogma, I have been unable to resist trying out new ideas or new technics.

It was my good fortune to have had unusually good teachers in grammar school, high school, college, and

medical school, and to have spent all my professional life in association with men who were testing new ideas about disease and its management. Perhaps I was fortunate, too, in never having had a fellowship or an assigned problem, but being just a doctor who looked into ideas which arose from practice.

Opportunity to look into such problems was always freely granted. Professor Eugene Opie let Mr. Asami and me study heteroplastic bone formation in the laboratory at Washington University; Cyrus Sturgis gave Tinsley Harrison and me a chance to measure blood flow in rabbits before and after inducing pneumothorax. At Stanford the generosity of Doctors Thomas Addis, Paul J. Hanzlick, and Emile Holman made possible studies on dogs, rabbits, and rats, on digitalis, hypertension, renal and hepatic metabolism, and on the mechanism of proteinuria In Brooklyn, Doctors Jean Oliver, Caspar Burn, and Patrick Fitzgerald enabled me to study the coronary arteries of infants and the heart valves of adults, while Doctor Edward Muntwyler gave me a chance to see what happened to sodium when cationic exchange resin was ingested. I can think of no one who has had so many generous and long-suffering colleagues, no one who was a guest worker for so long before becoming a guest lecturer.

In medical school, it was obvious to me that among my teachers many, like James Herrick, Leo Loeb, Frank Wilson, and Dallas Phemister, had minds far keener than mine. Two of them, Eugene Opie and Joseph Erlanger had made great original contributions while medical students. Because I did not realize that a tremendous

expansion of investigation was under way, which would give every dub a chance in the laboratory, it did not occur to me to try to become a teacher or investigator. After completing my residency I was grateful to Doctors A. C. Reed and Réné Bine for letting me get started in practice as the "boy" in their office, which commanded a fine view of a lovely park and the San Francisco hills. A year later, Professor Arthur Bloomfield astonished me by an invitation to join the group at Stanford Hospital and use the facilities of the laboratory for clinical physiology which had been organized by Albion W. Hewlett. Such an invitation, like that which gives me a chance to join the Porter Lecturers, is doubly welcome to one who realizes that the honor is not deserved. He can only hope it will be justified by events.

Contents

The Clinical Significance of Pulsations Evoked by the Heartbeat

LONG BEFORE Cesalpinus or Servetus had their visions of the heart pumping blood into arteries and receiving it from the veins, it was known that pulsations of the arteries were due to contractions of the ventricles. Graphic recording of the precordial, the jugular, and the carotid pulsations was systematically made after 1865, and records of pulsatile motions of the entire body first were reported in 1877, but were not studied carefully until 1937, long after the peak of clinical interest in the local pulses had passed In the decade before 1905, carotid and jugular pulses were intensively studied in order to analyze the clinical irregularities of the heart, but when electrocardiograms made such study more elegant, recording the pulse practically vanished from the clinic. Only recently has it been rediscovered that some nodal or atrial arrhythmias can be analyzed only by means of jugular pulse tracings, which bring to light atrial contractions when the atrial *P* waves are lost in the ventricular complex

In the last decade the recording of pulses again has become a useful method of study in clinics where surgical intervention is part of the management of congenital or acquired disease of the heart and great vessels In most medical centers pulsations are recorded only from the inside of the heart or arteries, after catheters have been

introduced. When Forsmann described the ease with which he passed a catheter down his own veins, from elbow to right ventricle, he stirred only passing interest in the diagnostic possibilities. Thanks to Cournand, the method has now become a standard procedure, which provides pulse records from all the chambers of the heart and from the pulmonary artery and its branches.

Because the heart is so often explored surgically, direct *determination of the pressure pulses in the auricles* and ventricles has become a standard procedure in the operating room, while needle puncture of the left atrium and ventricle through the chest wall has been employed extensively in several clinics. The discomfort and risk to the patient of these procedures are no greater than those associated with some other diagnostic procedures, such as bronchoscopy and gastroscopy.

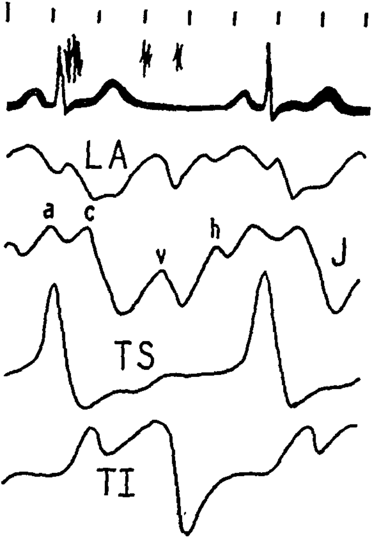
Many clinics which make constant use of cardiac catheterization fail to employ other methods or *recording* pulsatile phenomena associated with the heartbeat. However, some of these simple procedures may provide decisive information concerning heart disease with no discomfort to the patient, and with far less expenditure of time by skilled personnel. In our clinic, the recording of apex beats, jugular pulses, and pulsations of carotid, brachial, and femoral arteries is not routinely performed on every person suspected of having heart trouble, but one or more such records provide us with invaluable objective evidence in many cases.

With the roentgen ray one can observe and record the pulsations of the aorta, pulmonary artery, the atria and ventricles, with no discomfort to the patient. These

records (electrokymograms or slit kymograms) on occasion have provided the decisive datum in cases remaining obscure after study by other methods. During the seven years that we have been recording the pulsation of the *body (that is, the "big pulse" as distinguished from local* pulsations), we have found these traces, which Isaac Starr named ballistocardiograms, of great interest in all types of heart disease. They are of outstanding value in cases of coronary disease, and in studying the response of the circulation to exercise, smoking, and digitalis.

Local Pulses

(1) The Venous Pulse. The jugular pulse and the esophageal pulse obtained at the level of the left atrium give us the same sort of information about auricular activity as do the curves of motion recorded by the electrokymogram, or the curves of pressure recorded through the catheter (Fig. 1). In the jugular pulse, the a wave is due to atrial systole; c and v waves occur at the beginning and end of ventricular contraction. In curves of border motion, atrial systole produces a negative a wave, since atrial volume decreases, while in the jugular pulse the a wave is positive, because pressure rises in the atrium and vein. Both types of curve show a positive wave in early ventricular systole, the c wave. Then there is a sharp fall, due to the decrease in ventricular volume and pulling down of the A-V valves during systolic ejection. Atrial volume and pressure both reach their lowest levels during the middle of the ventricular systole. Then both begin to rise, because venous inflow reaches the highest rate noted at any time in the entire cardiac cycle. The late systolic rise



[illegible] the three heart sounds and [illegible]

in atrial pressure and volume continues into early diastole, with only a slight fluctuation as ventricular systole ends and the semilunar valves close.

When pressure in the relaxing ventricle falls below that in the atrium, the rise in atrial volume and pressure *ends abruptly, at the v peak, and both fall rapidly as* blood passes into the ventricles. In healthy young people and in young dogs, the atrial pressure falls to the ventricular diastolic level in a few hundredths of a second, and then there is a sharp rise in atrial pressure and volume reaching a peak, *h*, within 0.12-0.16 second after the second sound (Fig. 1, LA and J). During the middle of the intersystolic period, in these healthy young subjects, there is no further change in ventricular volume, and the wave of pressure, which caused the early diastolic rise in atrial volume and pressure, passes out into the cavae and the pulmonary veins. Fifty years ago Hirschfelder and Gibson noted the jugular *h* wave as the invariable accompaniment of the normal third sound, and ascribed it to reflux from a rapid ventricular inflow. This wave and the retraction of the apex which occurs at the same instant vary with respiration. The sharpest apical retraction and the tallest *h* waves occur with the loudest third sounds in each respiratory cycle As we grow older, this phenomenon disappears, for the pulmonary veins and atria are less elastic and empty more slowly in early diastole.

In heart failure, the v peak is sharp and the early diastolic emptying of the atria is swift, because the veins are dilated and tense If there is right heart failure, the jugular h wave may reappear, together with apical retraction and a third sound, or proto-diastolic gallop. When

an atrio-ventricular valve becomes insufficient with high pressure in the ventricle, there is little or no fall in the venous pulse after the c wave, and eventually there is a rise through systole and into early diastole. With stenosis of an atrio-ventricular valve the venous pulse shows a very slow fall after the *v* wave in early diastole. This fall is unusually abrupt and deep in cases of constrictive pericarditis or of insufficiency of an A-V valve, and is followed by a very abrupt rise synchronous with a gallop. The lesions which produce these changes in the venous pulse can usually be diagnosed without having phlebograms, just as pulmonary tuberculosis can be recognized without chest X-ray films. But we think it is wise to look at chest films in patients having pulmonary disease, or at pulse curves in any case where the therapeutic program depends on complete and accurate diagnosis of the physiologic state of the heart.

(2) *The Arterial Pulse.* Whenever there is any suggestion of aortic valvular disease, it is useful to record the carotid pulse. When there is a suspicion of pulmonic valvular disease, the electrokymogram of the pulmonic artery is most informative (Fig. 2). Sharp upward slopes, beginning soon after the Q wave, and rapid collapse in early diastole are characteristic of high stroke volume or valvular insufficiency; slow, delayed upstroke and late peak on the arterial pulse wave are characteristic of low stroke volume or valvular stenosis. In emphysematous old men the diagnosis of aortic stenosis or insufficiency may be proved by pulse waves even though the heart sounds are distant and no murmur can be heard. In one cyanotic cardiac child, diagnosed as having tetralogy of

Fallot by an expert, finding large normal pulses in the slit kymograms of the aorta and pulmonary artery led to the correct diagnosis of transposed great vessels.

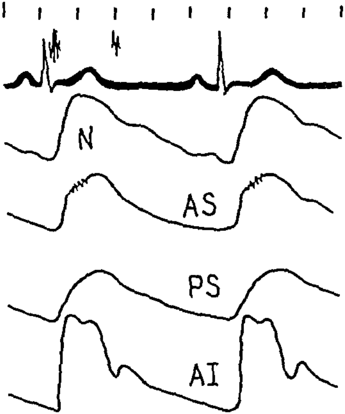
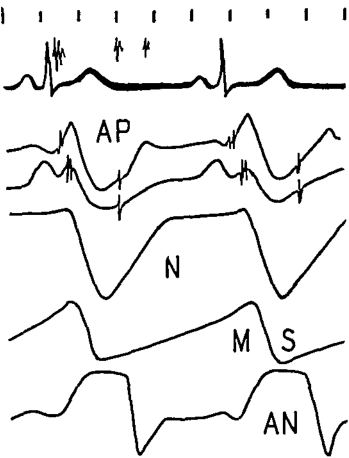


FIG. 2. Electrocardiogram and first and second heart sounds at top, time in 0.2 second. Normal [illegible] valvulotomy.

A comparison of carotid and femoral pulses, inscribed with the electrocardiogram, allows one to demonstrate vascular rigidity as shown by rapid rate of progress of the pulse wave. It also provides evidence of aortic narrowing, by coarctation or thrombosis, since such lesions reduce the steepness of the wave front and delay the peak of the wave in reaching the groin.

(3) *Cardiac Pulses.* In 1875, Galabin concluded that the apex beat was not due simply to motion of the ventricle as such but that certain features must be due to "*some coincident occurrence, and this can only be the* locomotion of the heart as a whole." It is now known that sharp deflections synchronous with the heart sounds or gallop sounds are actually due to concussion—that is, to the shaking of the whole heart by vibrations of audible frequency, and not to thrusts. Also some of the thrusts are not due to local bulging; some of the retractions are not due to local contraction of the ventricular walls, but are due to "locomotion of the heart as a whole." This locomotion is due to recoil of the ventricles from the jet of blood during ejection, or to impact of blood on the atrioventricular septum or on the bifurcation of the pulmonary artery. Such forces move the whole mediastinal mass. Transmitted to the thorax, they produce the motions of the whole body which are seen in ballistocardiograms. It is often forgotten that these forces move the human heart, which weighs less than 1 kgm., much farther than they move the whole body, which weighs over 50 kgm.

All the pulsations which are described as "typical" of the normal or of some form of vascular disease show considerable variation from one subject to another, and the


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normal passes into the abnormal by imperceptible stages, just as do the electrocardiographic ventricular complexes. Also, combined lesions are frequent, and many subjects with aortic valvular disease show slow rise of the carotid pulse, due to stenosis, and rapid fall, due to regurgitation. Thus the curves in Figs. 1-3 are only intended to show how disease may affect the curves. The greatest variation occurs in the apex traces, which vary from point to point in the same interspace of every subject. Because of difficulty in preventing carotid pulsations from affecting the jugular trace, the latter also changes with different applications of the pick-up.

The apex beat, precordial beats recorded at other sites, and motions of the ventricular border recorded by roentgen technics no longer can be accepted as reflecting the motion of the ventricular wall with reference to the center of mass of the heart. Yet these records may demonstrate paradoxical motion due to local injury (Fig. 4), they may show calcification in valves, the annulus, or the pericardium, and they may accurately mark the instant when mechanical systole begins. Several times we have recorded slow filling of the left ventricle as well as calcified mitral valves in older patients with no diastolic murmur or other decisive evidence for a stenosis of the mitral valve.

The methods of recording motion of the roentgen shadow include cineradiography and -fluorography (that is, 8 to 30 roentgen films of photographs of the fluorescent screen per sec.); angiocardiography, with contrast material introduced to outline chambers and blood vessels; electrokymography (motion of points on the border, or

density of a point over the heart or lungs, recorded like the electrocardiogram), and slit kymography. The latter is the simplest, requiring normal roentgen methods and a device to move the film across slits in the lead screen for 1 or 2 seconds. In our tricardiograph a 6½×8½-inch film records motion at 18 points on the left heart border for 1.3 seconds In the outer upper corner of the film, the electocardiogram and ballistocardiogram are recorded for 6 seconds, the roentgen exposure beginning after 4 seconds and marked on the electrocardiogram. The cycle recorded in Fig. 4 was a normal sinus beat, but an ectopic one occurred two cycles before. Unless the electrocardiogram and ballistocardiogram are thus inscribed, the border motion may deal with an ectopic beat, or one of small volume because of a Valsalva maneuver by the subject. The latter is revealed by the ballistocardiogram. In the tricardiogram one knows that a normal cycle or an abnormal cycle was recorded

In Fig 4, frame 1 is an aortic area; frames 3 to 11 the left ventricle from base to apex. Frame 9 is frankly paradoxical, bulging as the basal part moves in during systole Frame 8 is also paradoxical, 7 and 10 transitional zones with minimal motion, 11 at the apex, shows the "kick" at the onset of the systole. Diastolic filling is rapid (frames 4-6), as is usual in all types of myocardial failure and in constrictive pericarditis

The Big Pulse, or Ballistocardiogram

The whole body shakes with each heartbeat, as one can readily observe when one stands on the ordinary

spring balance and watches the pointer. A patient with aortic insufficiency may cause visible shaking of a heavy bed at each heartbeat. The "big pulse" was first recorded by Gordon in 1877, and half a dozen men made casual observations using various technics over the next half century. But the story of the big pulse really begins with the work of Isaac Starr.

About twenty years ago Starr set out, like Columbus, to seek a short new approach to a very well-known ob-

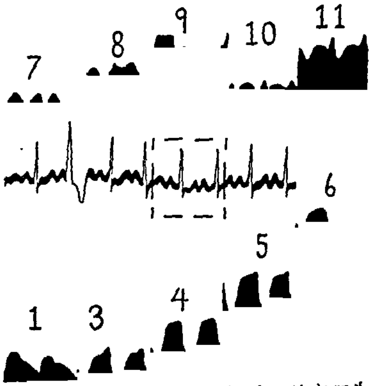


Fig 4 Ten frames of left border motion, drawn from a tricardiogram of a woman of 50 with hypertension, and heart failure, no angina or episode suggestive of myocardial infarction. Electrocardiogram normal except for deep Q in AVL. The dotted box shows the electrocardiogram synchronous with the recorded motion. Discussion in text.

jective. For fifteen years he sought in vain to estimate the cardiac output from head-to-foot motion of the body during systole. Like the famous Genoese he kept discovering new and useful territory in this unexplored physiological realm, and uncovered new methods of detecting clinical disorders of the circulation. He mapped out the main features of ballistocardiography. Those who followed filled in details and confirmed the accuracy of his landfalls.

It is quite clear that the most accurate record of the force imparted to the body by each heartbeat gives only a rough index of cardiac output in health, and goes very wide of the mark in heart disease. It gives a fair index of the kinetic energy imparted to the blood, in health, but since this is only about 1 per cent of the energy the heart liberates in overcoming arterial resistance, the ballistocardiogram does not tell us the force of cardiac contraction. In 1945, William Hamilton studied ballistocardiograms recorded in three planes, concluded that the method could not yield accurate quantitative information on cardiac function, but pointed out that correlation of the patterns with clinical conditions might be of value, as Starr's papers already had indicated.

Technics of Recording the Big Pulse

There is now a profusion of devices for recording vascular pulsations, from inside the circulation and from the surface of the body, and methods for recording the big pulse, or ballistocardiogram, are even more numerous and varied in principle In the records of pulsatile phe-

nomena with which medical students are familiar, one common feature predominates. The curves show the *displacement* in space of some object — an arterial or venous wall, the chest wall, a membrane (in a manometer), or a fiber or electron beam (in a galvanometer). In each case the displacement is resisted by the tension of the vessel wall, the membrane, the fiber, or the inertia of electrons moving at high velocity. In every case, one measures a force and the curves are calibrated in mm. of Hg (1330 dynes per $cm.^2$) or in millivolts.

Starr calibrated his instrument for recording the big pulse so that the curves could be measured in dynes. He measured the displacement of a platform held by stiff springs, which resisted the forces transmitted by the body to the platform. It is now known that the body, resting on the elastic cushions of the shoulder-blades, encased in muscle, and the buttocks, heavily padded with fat, moves on the Starr table 5 to 7 times farther than the table, restrained by its springs, with each ballistocardiographic wave. But as the springlike action of the cushions of tissue is relatively weak, the natural frequency of body motion is relatively slow, 4-7 cycles/sec., compared with that of the table with a 70-kg. dead-weight load (10-14 cycles/sec.). As a result of these conditions, the curve of motion of the Starr table is almost identical with that of motion of the body relative to the table, and it is altered by artefacts due to interplay between cardiac forces and afterwaves due to natural frequency of the body. Similar artefacts would occur in electrocardiograms if a loose and poorly damped galvanometer string were used.

These defects were well known to Starr, who pointed out that there was reason to doubt "if any type of ballistocardiograph gives a completely true account of the forces which originate within the body, and I shall cling to the simplest and most practical type, and learn what I can from it." It now appears that the simplest way to get curves almost identical with Starr's is to put a coil of wire on the shins, a permanent magnet close to the coil but resting on the table, and integrate the electrical signal, which gives velocity of motion, so that displacement is recorded by any electrocardiograph to which the coil is connected. Sadly defective from the standpoint of accurate recording, this instrument is a valuable diagnostic tool and may be compared with various reagin precipitation tests for syphilis, which are theoretically absurd but clinically useful.

If, instead of recording the displacement of a body restrained by very stiff springs, we record the acceleration of a body which is not restrained at all, we get identical curves Pressure, force, and acceleration are so related that the curve of acceleration of a free body, acted on by a given force, is the same as the curve of pressure when the same force acts on an immovable object, or the curve of displacement of the object restrained by powerful springs Thus, the true ballistocardiogram can be obtained either by restraining the skeleton with powerful springs and recording its displacement, or by having it free to move in space, and recording the acceleration curve The first ideal cannot be attained in practice, because there is a barrier of spongy tissue between the skele-

ton and the skin; the second because the body cannot be suspended freely in space.

Recording the acceleration of the body floating on a pool of mercury, or on a very light frame hanging from long cords, gives a reasonably close approach to the ideal curve of acceleration of the body floating freely in space, but these methods do not lend themselves to recording the big pulse in three planes, and usually only permit recording head-foot motion. It must be emphasized that *such acceleration curves have no relation to curves of acceleration of motion of the body relative to a solid platform or Starr table.* We can also, by electrical circuits, convert the ordinary pulse waves or electrocardiograms to curves of acceleration instead of displacement. This brings out a wealth of detail in the curves, since every plateau becomes a dip to the base-line, every notch a deflection past the base-line. The curves of motion of the shins, picked up by the coil-and-magnet technic, are easily differentiated to acceleration signals in which change due to age or disease is strikingly evident, but such curves are entirely different from those recorded by accelerometers on the body free to move in space.

In our laboratory we used the simplest technic, recording displacement from the shins, for a screening test. Acceleration of the body lying on a 2-kg. frame hung from cords 2 m. long is used as a reference in evaluating other technics for recording head-foot forces, using the instrument made available by the generosity of the Sanborn Instrument Company. All our serious work is done with a home-made device for recording, in three planes, *the forces acting on the thorax,* which is restrained by powerful springs in all three planes. Here the natural

frequency of the loaded instrument is 10-15 cycles/sec., and the head-foot trace closely resembles that of acceleration of the body on the Sanborn instrument (Fig. 5).

In relation to records of local pulses, it was noted that these may vary when points close together on the surface of the body are studied. When motion of the head and shins is recorded simultaneously, it usually is apparent that in late systole the shins move footward much more than the head. When we record the motion of the freely moving body, pulses as far apart as the arch and the bifurcation of the aorta are summated. Partly because we are closer to the generator of the big pulse, and partly because the thorax is the least tremulous part of the body in our shaky old subjects, we find records made from the thorax more satisfactory than those from the low-frequency-acceleration device even for recording in the head-foot plane.

With any system of ballistocardiography, a simultaneous trace of the electrocardiogram, heart sounds, or arterial pulse is necessary so that the timing of the waves can be studied. Multichannel direct-writing galvonometers, though costly, are now available in most laboratories where heart disease is under investigation Our home-made screening device cost 0.3 per cent, our home-made three-plane pick-up cost 2.5 per cent, as much as our four-channel galvanometer. So much for the technical and financial background of recording the Big Pulse

The Ballistocardiogram, or Big Pulse, in Health and Disease

The three-plane traces of the ballistocardiograms of healthy young adults are more uniform than their electro-

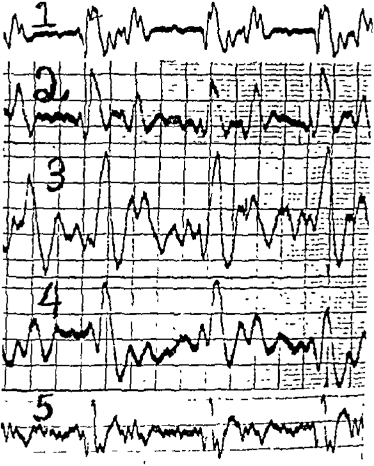


Fig 5 From a 55 year-old normotensive man, with no history or physical evidence of heart disease All the traces show head-foot ballistocardiograms, and the central beat in all has the same relation to simultaneous electrocardiograms 1, body acceleration from a light, very low-frequency stretcher (Sanborn) Note the detail and the constancy of this pattern. 2, from our three plane system, displacement of a spring-opposed high frequency platform pressing the shoulders footward with a constant force of 10 kg with the feet against a solid support. Note that the peaks of displacement of the firmly re-

cardiograms in the three Einthoven leads When the system is adjusted to give equal signals when equal forces are applied in each plane, the systolic waves in the head-foot traces are three to five times larger than in the other two planes. The diastolic waves, relatively small, are most evident in the lateral trace. The capital letters *H*, *I*, *J*, and *K* were introduced by Starr to identify the waves evoked

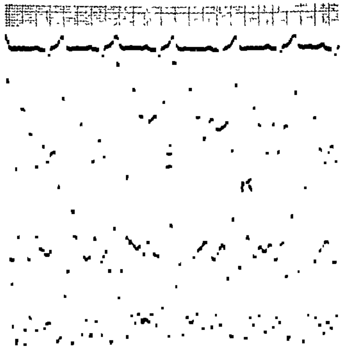


FIG 6 A normal three plane ballistocardiogram, with lead II at top, next the head foot (upward on the trace is headward), next the lateral (upward is leftward), and at bottom, the dorsoventral (upward is backward motion) The [illegible]

by ventricular systole, while *h*, *i*, *j*, and *k* were used by Nickerson for the waves due to atrial systole in heart block. During normal breathing, the *I-J* waves in the lateral plane are largest in expiration; those in the other two planes are largest in inspiration (*Fig. 6*). Pulmonary arterial flow is increased during inspiration, the pulse pressure in the pulmonary artery rising 30 per cent or more as compared with the smallest pulse in the cycle. Aortic outflow is increased about 10 per cent per beat during expiration. Thus, right ventricular force dominates the pat-

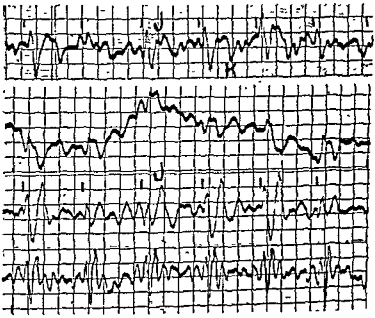


FIG 7 From a 70-year-old hypertensive, asymptomatic and leading a busy professional life Upper trace, head-foot inscribed from the shoulders, shows [illegible] Below, three-plane [illegible]

tern of the classical head-foot trace, and the lateral force varies with left ventricular ejection. When breath is held in full inspiration, the total ballistic force is large, because low intrathoracic pressure augments venous return. In deep expiration, held without closing the glottis, ballistic force is minimal, but the lateral waves are relatively large because the outflow tract of the left ventricle is tipped toward the horizontal by a high position of the diaphragm. The ballistic vector changes more than the vector of electrical forces during respiration.

In old people and those with heart disease, the pattern of the big pulse changes far more radically than does the ventricular electrical complex. While some octogenarians have ballistocardiograms indistinguishable from those of youths, most people over 60 show far more variation in head-foot waves during a respiratory cycle, and relatively large right-left or front-back waves. In stoop-shouldered, deep-chested men, the head-foot trace may be small and chaotic, while a normal trace of large amplitude appears in the front-back plane (Fig. 7). In many healthy old people, the lateral waves are larger than head-foot waves, especially during expiration. This is even more frequent in older people who have recovered from bouts of heart disease and those who still are handicapped by such disorders. Some of the cardiacs show marked decreases in systolic waves in all three planes, especially during expiration.

Age seems to have no effect on the diastolic waves, but many forms of heart disease cause very great increase in the early or late diastolic motion. It is not unusual to see these diastolic waves dominate the entire pattern in all

sorts of cardiac patients. When the patient has gallop sounds in early diastole, it is practically certain that the big pulse will show a marked heave headward, rightward, and backward beginning with the sound. If there is a presystolic gallop, the large waves follow atrial systole (*Fig.* 8). But the thoracic pulsations may persist after the gallop has disappeared, or be present for a long time before it is heard. These diastolic waves may be exaggerated only during expiration, or after meals, or when the patient smokes. When digitalis is given, the disappearance of this wave may be the only objective proof that the drug has restored heart action to a more normal state (*Fig.* 9). Often an increase in the size and a reversion from an abnormal pattern of the systolic ballistic waves is associated with the disappearance or decrease in diastolic waves.

The normal systolic complex consists of a small wave (*H*) headward, rightward, and backward, followed by a larger one in the reverse direction, and then by the largest wave, *J*, headward, rightward, and backward. The peak of the *J* wave occurs about 0.2 second after the onset of

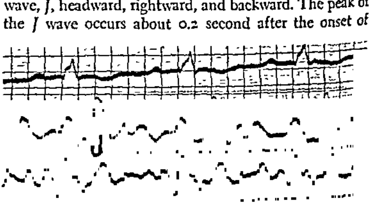


FIG 8 A 40-year-old woman with bundle branch block of undetermi[ned] origin There was a presystolic gallop. The *head foot trace shows a rather la[rge]* presystolic *H* and a small broad *I* wave, but in the lateral the sharp left r[ig]ht presystolic wave, which begins 0.10 after P, is the main deflection in ea[ch] cycle.

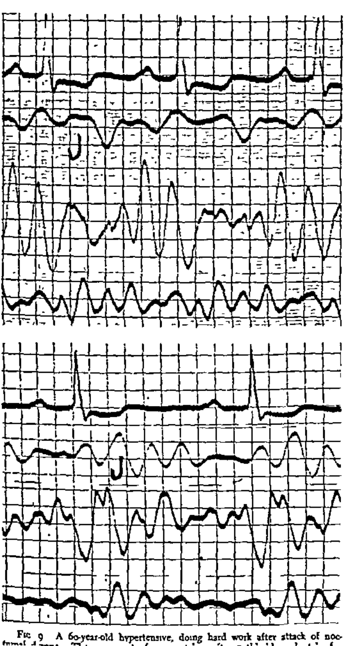


FIG 9 A 60-year-old hypertensive, doing hard work after attack of noc-

electrical activation of the ventricles. In traces from the shin or the Starr table a deep wave, *K*, occurs at the end of systole, but in lateral traces or in the acceleration trace there is a gradual return to the base-line. In heart disease, *I* becomes shorter, *K*, deeper; and the *J* wave may be notched. Maximum abnormality, rarely seen except in hearts badly damaged by infarction, produces a plateau with three ripples in early systole when the *H* wave is fused to a notched *J* (Fig. 10). This is followed by a deep *K*, and by a large headward and rightward wave, *N*, in early diastole.

It is generally agreed that the main systolic waves are due to forces generated by recoil from ejection of blood, or by its acceleration and deceleration in the aorta and pulmonary artery. In the head-foot trace made by Starr's technic, the height of the *I-J* slope is proportional to the square of the velocity of flow in the great arteries, but in disease and in age this relationship in greatly altered. The normal and abnormal waves which follow auricular systole and early diastolic ventricular inflow apparently are related to the velocity with which ventricular filling begins and ends. However, the main diastolic waves are not directed footward and leftward, along the axis of the inflow tract, but in the opposite direction. These large waves begin about 0.15 sec. after the *second sound or the* onset of the auricular *P* wave, and their onset is synchronous with the gallop sound and the apical retraction *which coincides with the end of* rapid ventricular filling.

The probable explanation for this phenomenon is that blood rushing through the funnel of the A-V valves spreads through a wide arc and the force is thus not ap-

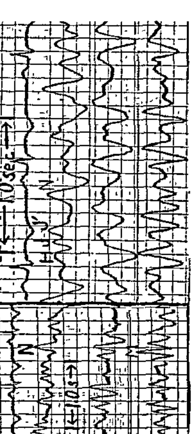


Fig 10 From a 55-year-old man, convalescent eighteen days after a myocardial infarction The left shows one respiratory cycle, with the three planes of the ballistocardiogram, the right, two heart cycles during held respiration, the first cycle showing the classical pattern of fused *H-J* with notched *J*, seen in cases of severe damage from recent infarction. Note that the large *N* wave in head-foot traces is protodiastolic, while the large lateral and dorsoventral traces are presystolic

plied effectively in any one vector. But the reflux at the end of filling is focused by the concave ventricular wall on the A-V orifice and is applied with great force as the valves are pushed into the atrium. In cases where large *N* or presystolic *H* waves are found, studies of apical and ventricular border motion often show that the damaged heart fills faster than it discharges during systole. In any event, we can accept these large diastolic waves in the big pulse as evidence that pressure is high in the inflow tract. Just as a high-blood urea is the index of failure of the kidneys, high venous pressure is the index to failure of a ventricle. Right ventricular failure can be estimated by measuring pressure in the systemic veins, but the large diastolic waves of the big pulse may provide early and striking evidence of left ventricular failure, which is the type most often present in patients with hypertension, coronary disease, or aortic valvular disease.

In heart failure with large diastolic ballistic waves, the systolic waves may be small and bizarre or may be normal. In heart failure with anemia, acidosis, hypermetabolism, arterio-venous shunts, or leaking heart valves the systolic waves may be very large even in advanced failure. Just as blood urea may rise when the kidneys are excreting, and the body manufacturing, two or three times the normal basal level of this product of metabolism, so the heart may fail when the ventricles are moving two to six times the normal basal flow of blood per minute. The normal person on exercise will move a similar amount of blood but shows little rise in venous or in end-diastolic ventricular pressure. In heart failure these pressure levels may be several times normal, and the rise, on increasing the

venous return by exercise, is much steeper than in normal persons. Thus, mild exercise may improve the systolic ballistocardiogram while bringing to light abnormally large diastolic waves. When the myocardium is severely compromised, exercise causes not an improvement, but a deterioration, in the systolic pattern.

When the history and physical findings point to congestive heart failure, the presence of large systolic I-J complexes in one or more planes immediately raises the question as to whether the patient has one of the disorders which cause a high minute-volume flow through one or both ventricles. Auscultatory and other signs may show that one or more valves are insufficient, or that a congeni-

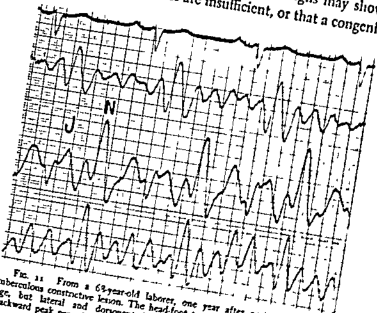


FIG. 11 From a 63-year-old laborer, one year after pericardiectomy for tuberculous constrictive lesion. The head-foot trace is normal and large for this age, but lateral and dorsoventral traces show large protodiastolic waves, backward peak precedes the lateral by 0.05 sec.

tal or acquired shunt is present. Hyperthyroidism, leukemia, or Parkinsonism may have raised heat production, or anemia, acidosis, or thiamine deficiency may have evoked peripheral vasodilatation. Correction of some of these causes for high output may decrease the systolic waves and relieve the patient of his distress. Some of these disorders are obvious, others must be sought for, and ballistocardiograms may show the need for such a search when high output has not been suspected.

In many cases, the abnormal diastolic waves are especially striking, and in some cases they are only evident, in the right-left trace (Fig. 11). The reason for this presumably is that the inflow tract from the atria is usually much more transversely placed than the outflow path through the semilunar valves. In cases of heart block, the auricular complexes of the big pulse are usually very much larger in lateral than in head-foot traces. In early ectopic beats, where the head-foot ballistic waves are small, the lateral ones may be large (Fig. 12). An ectopic beat, occuring before the ventricles have refilled, fails to eject blood into the arteries, where diastolic pressure is still high, but it can always halt inflow, and force the A-V septa toward the atria. Usually, the large lateral wave with such a beat occurs late and represents a gallop wave. Such a wave may be clearly evident in the jugular veins, as well as in the lateral ballistocardiogram, during a ventricular ectopic beat which causes no second heart sound, arterial pulse wave, or head-foot *I-J* complex.

The most modern and complex technics for inscribing the acceleration curves make comparison of one patient with another more dependable than when the Starr table

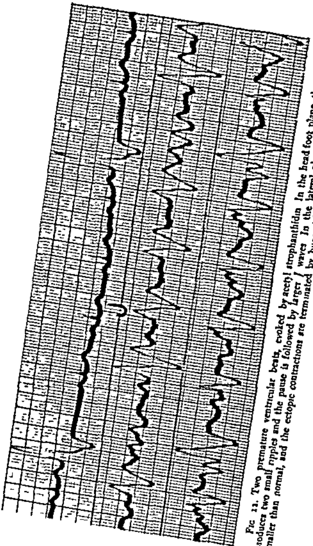


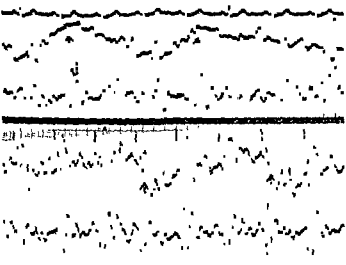
FIG. 12. Two premature ventricular beats, evoked by acetyl strophanthidin. In the head-foot plane, the early beat produces two small ripples and the pause is followed by larger J waves. In the lateral plane the post-ectopic H-I is smaller than normal, and the ectopic contractions are terminated by huge right ward waves.

is used, and bring to light small but significant waves in the big pulse. However, the curves inscribed from the shins or the Starr table often show greater variation in the patterns inscribed by any given subject as a result of respiration, exercise, nicotine, or digitalis. The natural frequency of the unrestrained body lying on a platform, is very close to the frequency of the ballistic waves, and the curves inscribed from such a system represent interplay of the imposed forces and the natural oscillations evoked by the initial wave in each cycle. It requires only a small change in the amount or duration of any force applied to the body to modify the phase relations between natural and ballistic oscillations. The Starr technic provides a very sensitive indicator (Fig. 13) of change in pattern, of any given subject from beat to beat, but it is less satisfactory than the acceleration type of curve for timing the events of the cardiac cycle or comparing one person with another.

It is worth noting that the effects of smoking on the ballistocardiogram, which provide the most sensitive and dependable objective evidence of coronary disease, were missed by those who used elaborate technics and were brought to light by the simple pick-up on the shins. *One of the most impressive findings with the* ballistocardiograph is that normal traces, under basal conditions, are found in a higher proportion of men who have had complete clinical recovery after myocardial infarction than in those who have angina of effort but never had an episode of infarction clinically. During attacks of angina, there usually are transient but marked changes in the ballistocardiogram even when pulse rate and blood pressure

change scarcely at all, and even when the electrocardiogram does not alter during the attack. These are the facts which support Starr's view that head-foot traces, recorded with the simplest device, provide evidence of abnormal reaction of the heart which corroborate clinical evidence even though all other objective tests remain unchanged.

In all types of physical examination or laboratory testing, it is not so much the deviation from some ideal normal standard which is important in judging the condition of a patient as the rate and direction of change. A sys-



[illegible] a 40-year-old [illegible] twenty ye[illegible] cycles wh[illegible]

tolic murmur, a curious blood picture, or a roentgen shadow found in a person who feels well may be significant if it becomes more striking as time passes or if symptoms appear. If it remains the same, or gradually disappears, it has only historical significance. An apparently normal trace of the big pulse which deteriorates on exercise, during a spontaneous bout of chest pain, or on smoking a cigarette is of far more significance than a very abnormal trace which is constant under all these circumstances A simple method of recording these changes is of more value to the clinician than a bulky or complex device that yields traces, which, though superior from the standpoint of the mechanical engineer, require close scrutiny or measurement in order to detect changes which are obvious at a glance with the other method.

When a patient exhibits an illness compatible with coronary disease and has changing electrocardiograms from which one can deduce the site of myocardial damage, the chief value of the big pulse is in the case where its waves show a normal pattern. While the threat to life from subsequent attacks can never be accurately guessed from any of these tests, the man with the normal ballistocardiograms has less risk of having angina or heart failure than the one with poor traces. On the other hand, if a man in apparent good health exhibits bad records while smoking (Fig. 14), it may be wise to give him the same advice about smoking and diet which one would give to a person who had completely recovered from an actual infarction. If a hypertensive in relatively good health has a fairly good ballistocardiogram but the trace becomes even better when the patient is taking digitalis, it would

be wise to keep him on digitalis as prophylaxis against heart failure (Fig. 9).

The great objection to recording the big pulse, which one hears at every panel discussion, is that doctors are creating invalidism by misreading the traces. Most of the men who offer this criticism are the same people who, for many years, have put to bed for six weeks anyone whose electrocardiogram could be interpreted as evidence for myocardial infarction. They are lineal descendants of the great London clinicians who aroused the ire of Sir James MacKenzie because they regarded systolic murmurs or even sinus arrhythmia as evidence for serious

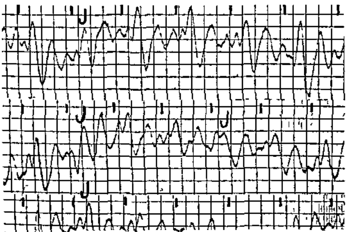


Fig. [illegible] Before [illegible] after three days [illegible]

organic heart disease, requiring rest and drugs. The pessimistic, timid, sadistic, or venal physician can create invalidism without any gadgets at all—he merely misinterprets the history.

In our experience, the traces of the big pulse have not been harmful to any patient, and they were helpful in demonstrating to some that smoking affected their hearts adversely and to others that the pumping function of the heart was unaffected by old or recent myocardial infarction. Like the automobile or the stethoscope, the ballistocardiograph can be deadly in the hands of careless operators, but is useful to the competent, cautious, and optimistic physician. By bringing doctors back to thinking of the heart as a pump, and not just a source of sounds and electrical currents, recording the various pulses evoked by the heartbeat and studying their alterations in disease or as a result of therapy should do more good than harm. We believe that there are far more good physicians than fools licensed to practice medicine, and simple tools which provide additional objective data about the circulation can be valuable to the good physician.

The Production of Sounds by Normal and Diseased Hearts

ALTHOUGH WILLIAM HARVEY noted that the heart made a noise with each beat, nearly two centuries passed with no further attention being given to this by physicians. Early in the nineteenth century, the French physicians who had revived Auenbrugger's discovery, percussion of the chest, began also to palpate the precordium and to listen to the sounds which accompany breathing and the heartbeat. This was done by the effective but awkward method of applying the ear directly to the chest, or putting a sheet of fine paper over the region to be ausculted. The esthetic barrier to ascultation was overcome when René Théophile Hyacinthe Laennec discovered that even a roll of paper or a wooden cylinder could be used to transmit sounds from the chest to the ear.

Laennec was recognized as a brilliant clinician before he made this observation, and he soon crystallized his experience with pneumonia, tuberculosis, and heart disease in a classical treatise which brought auscultation into world-wide attention. His contemporaries and elders in Paris were not impressed. The celebrated physician Broussais was outspokenly hostile to auscultation, but younger men from many lands eagerly mastered the use of the stethoscope. They soon corrected the minor errors in Laennec's original conceptions of the heart sounds, and by 1830 it was generally accepted that the second sound was due to sudden tensing of the semilunar leaflets

as the valves closed *at the end of systole. There was dis*pute, which still goes on, about the origin of the first sound. One school favored a purely valvular origin, similar to that of the second sound, but due to tensing valves at the atrioventricular orifices as systole stopped inflow to the ventricles. Another favored a purely muscular origin, due to tensing of the heart muscle. Most authors accepted the genial British compromise—the first sound had both valvular and muscular elements. Thirty-five years ago, when I was introduced to the heart sounds, any internist worth his salt could listen to a heart and announce "the valvular element is normal, but the muscular element is poor" or vice versa.

During the previous half century Potain and his pupils had made the French- and Spanish-speaking doctors acutely aware of the sounds in diastole which occurred with failure of the myocardium, but in the English-speaking world these sounds, and the significance of changes in intensity of the first sound, were either ignored or passed over lightly. Murmurs were the abnormality sought by most of my early teachers. The *Osler* of 1920 has "galloping consumption" but not "gallop rhythm" in its *index*.

With the use of phonocardiographs, which at first recorded sounds well and murmurs rather poorly, the attention of investigators became focused on "sounds." And now the sound underwent a mysterious transformation—it often became inaudible! Low-frequency vibrations of low intensity can be heard when isolated in complete silence But the human ear fails to note such waves when they occur immediately before or after a loud sound of higher frequency. On the phonocardiographic trace the

second sound may be prolonged or split, but it begins and ends abruptly. The first sound at the apex is quite different. Its large high-frequency oscillations, even when prolonged or split, stop and start abruptly, but they are usually preceded or followed by small waves of low frequency. Students of such traces concluded that the first sound had four elements, not acting synchronously like four stringed instruments giving the same note in four different octaves at the same time, but occurring in sequence with a total duration which could be three or four times that of the main high-frequency waves. The early writers who spoke of two elements had thought of them as simultaneous, and when the old doctor distinguished muscular from valvular, he was like a conductor who hears a note from the oboe given out simultaneously with the same note from the viola, and can tell whether either is off pitch, or too loud or too soft. From the descriptions of the four elements of the first sound, a clear-headed novice might easily believe that a good man could note abnormalties in any of these four elements. Fortunately, none of the authors of these descriptions ever drew that conclusion; most of them were aware of the fact that all but one element fell below the auditory threshold at any one site in most patients' chests

In people with normal atrioventricular conduction and sinus rhythm, a brief, low-pitched, inaudible oscillation, the "first element," is present just before or after the QRS of the electrical activation wave of the ventricles starts This is not present with auricular fibrillation, and with prolonged A-V conduction it is inscribed before ventricular activation begins. Thus the first element results

in part from atrial systole. This auricular oscillation may be detected by the ear if it is separated from the loud vibrations of the first sound by 0.04 second of silence. The longer this silent interval, the more audible such oscillations become. The audible presystolic sound has its own names: fourth heart sound, if detected in a normal subject; presystolic gallop when associated with heart disease.

In atrial fibrillation, there may be 0.02 to 0.04 second of low-pitched, low-amplitude oscillations before the loud vibrations of the apical first sound are registered. This feature of the phonocardiogram is usually called the muscular part of the "first element." It has nothing to do with the "muscular element" of the pre-phonocardiographic authors, who thought that in many people most of the audible vibrations, or according to some authors, all the audible vibrations of the first sound, were muscular. This brief oscillation is most readily demonstrated in mitral stenosis with auricular fibrillation, since it is not lost in the atrial element, and the main apical vibrations of the real first sound occur later than usual. Whether these oscillations are due to the apical thrust, with friction or impact of the heart or pericardium against the chest wall, or whether they actually arise in the myocardial fibers, has not been determined. Presumably the normal first element contains waves of auricular and ventricular muscular origin; actually all this is inaudible.

The second element of the phonocardiographers is the real first sound. In mitral stenosis, or after violent exertion, or in severely anemic people, this sound may be heard at a distance from the chest, and of course without using a stethoscope. It occurs 0.04 second after *Q* of the

electrocardiogram in nearly all normal people, but the interval may be more than 0.08 second in mitral stenosis. At the apex it may be split, or broadened, by asynchronism of occurrence of elements coming from the two halves of the heart. Although debate about the origin of these vibrations continued up to the year 1954, the recent studies on mitral stenosis have eliminated any possible grounds for doubt that this sound is due to "sudden tensing of the auriculo-ventricular (mitral and tricuspid) valve segments *and their chordae tendineae*" (*to quote* Byron Bramwell, writing in 1884).

The history of the interminable fight to place the origin of this sound in the heart muscle is of interest to students of human faith The most determined proponents of this view were men trained in Central Europe, and physicians who took as their gospel all German medical literature. This school was firm in the faith even after it had been pointed out that the intensity of the first sound was greatest if it occurred 0.02 to 0.14 second after atrial systole or rapid early diastolic filling had begun, and was least if it occurred 0.2 second or more after these events. Rapid inflow was known to take the slack out of the A-V valves and chordae tendineae by pushing atrial contents toward the apex Proponents of a purely muscular origin of heart sounds were not impressed by the fact that the first sound was very loud for a longer time in mitral stenosis, when the chordeae and valves are under pressure from the auricle much longer than in normal persons.

Advocates of the muscular origin of the first sound scoffed at records showing the disappearance of heart sounds in the exposed hearts of dogs when the heart was

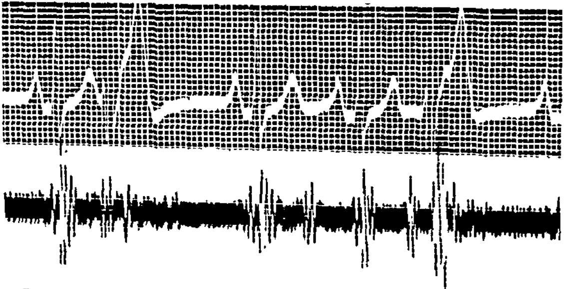


FIG. 15. From Lewis and Dock. The first ectopic beat is silent; the second causes a first sound louder than that with normal beats; neither opened the semilunar valves (no second sounds). The low-amplitude, low-pitched oscillations which precede the normal first sound and occur with the first ectopic beat were not audible. The first ectopic beat begins with the second sound; the second one begins 0.10 second later.

empty or the great vessels and atrioventricular sulcus compressed by a ligature. By applying a diaphragm to the surface of a dog's heart they could always get mechanical oscillations in such experiments and thus have an excuse for rejecting those done with technics, introduced by Wiggers and Dean, which avoided mechanical impact or friction on the microphone.

They ignored records of silent ectopic beats when electrical activation began 0.02 second after the second sound, and similar beats with very loud first sounds, but no second sounds, when activation began only 0.08 second later. Both ectopic beats might be due to the same type of electrical activation, both so feeble that the semilunar valves did not open. In the earlier beat, the A-V valve had not moved down from its position in midsystole, and no sound was produced. In the later one, inflow to the ventricles was at peak velocity. Although ventricular filling was far from complete, the valves and chordeae, pushed ventricle-ward, were slack when the ectopic beat pushed the valves into the atria and drew taut the fibers, making a sound louder than that due to normal beats (Fig 15). Fortunately, very early ectopic beats were rare, so this striking phenomenon could be left out of consideration by those who had faith that the valves were silent or soft-spoken, and only the muscle really sang out loud and clear.

One of the groups who cherished this faith observed, in the early 'thirties, that the first sound was late in mitral stenosis. This awkward fact is apparent in their studies on intensity of the first sound, and was mentioned to visitors to their laboratory. The first publication on late first

sounds in mitral disease came from one of these visitors, after waiting in vain for his hosts to discuss this phenomenon. In the last three years, a relation between delay of the first sound and the height of atrial pressure (or the tightness of the orifice) was brought to light by mitral surgery and measurements with the intracardiac catheter. Finally, the beautiful observations of Braunwald and his colleagues at Mount Sinai Hospital made available simultaneous left atrial and ventricular pressure curves which showed that in normal subjects the left ventricular pressure in early systole begins to rise above atrial pressure 0.03-0.04 second after the Q wave, while in mitral stenosis pressure between the two chambers is reversed much later, and the delay is proportionized to the tightness of the orifice and the elevation of atrial pressure. Kelly, Lyons, and our surgical colleagues have confirmed this at our institution. The comparison of time of onset of the first sound and the instant when left ventricular pressure rises above left atrial pressure leaves no doubt whatever that this sound occurs only when the mitral leaflets are forced toward the atrium.

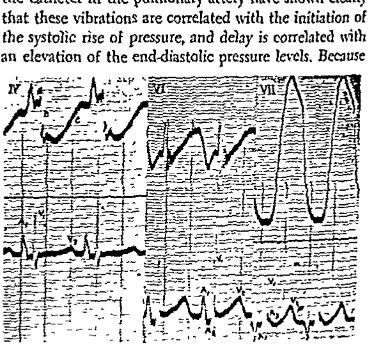
As noted above, the "muscular element" of the first sound complex on phonocardiograms is readily found in mitral stenosis; the oscillations are neither large nor audible. Ventricular pressure goes far above that at which the first sound occurs in normal people before a sound occurs in those with mitral stenosis. Thus, all those who ascribed the first sound to "tensing" the muscle find themselves unable to explain why no sound occurs when higher tensions develop in this form of valvular disease and until the mitral valve finally is drawn taut.

It is striking that there is no splitting of the first sound at the apex in mitral stenosis, owing to the normal vibrations set off by tricuspid closure and tension. The reason seems quite clear, however, if one recalls that patients with systemic arterial hypertension often have faint first sounds, and that in mitral stenosis the rise in pulmonary arterial pressure is far greater, in relation to normal levels, than is ever seen in systemic arterial hypertension. Ventricles which empty with difficulty are overfilled at the end of diastole and the A-V leaflets have no slack to take out when systole sets in. This seems to explain why the first heart sound in mitral stenosis contains no tricuspid element and therefore is not only loud but snapping It is never prolonged or split by asynchrony of elements from two sides One side is mute, the other is late, but loud and sharp

This discussion of the first sound has dealt particularly with what is heard when the stethoscope is applied at the apex, or point of maximum impulse. Over the great vessels at the base of the heart, a third element of vibration can be regularly inscribed with the phonocardiograph This may be continuous with the final oscillations of the true first sound, and it may be more intense than that sound in the region close to the clavicles. When there is pulmonic or systemic hypertension, this sound is delayed and accentuated Then its vibrations are heard as a distinct sound, to which the name "systolic click" is now applied In myocardial failure, when the second element is faint and pulmonic pressure is high, it is the third element which forms the largest waves in records from Erb's point, in the mid-precordium to the left of the

sternum, or even in records from the apex where the second element may be faint. The third element is properly called the vascular element, for it occurs because the fibers of the aorta and pulmonary arteries experience a sharp rise in tension when blood spurts into them in early systole.

These vibrations mark the onset of systolic ejection; they occur 0.08 to 0.12 second after the Q wave, with a delay when diastolic pressure is high, especially in the pulmonic artery where diastolic pressure may reach four to six times its normal level. The pressure records from the catheter in the pulmonary artery have shown clearly that these vibrations are correlated with the initiation of the systolic rise of pressure, and delay is correlated with an elevation of the end-diastolic pressure levels. Because



the third element is relatively high-pitched and rarely is loud at the apex when the first sound is loud, it does not give the impression of a split first sound. At Erb's point or the pulmonic area, it may give that impression, although the experienced examiner registers it as a click, not a sound or a snap, because of its pitch.

The vibrations which show up on phonocardiograms after the click ("third element"), are called by Rappaport and Sprague the "fourth element" of the first sound. These appear to be very low-frequency oscillations, and are very feeble. Obviously inaudible, these jiggles may be due to motion of the precordium as the heart moves against it during the most rapid changes of ventricular volume or to mediastinal motion due to ballistocardiographic forces.

When MacLeod and Cohn registered the pressures in the right and left sides of the hearts of dogs, using a piezo-electric pick-up on a rod introduced through the carotid artery or the jugular vein, they recorded at the tricuspid orifice rapid vibrations synchronous with the first sound (Fig. 16). At the root of the aorta they recorded intense vibrations with the second sound, also rapid but less marked vibrations synchronous with the second and third elements of the first sound (Fig. 17). It is worth emphasizing that these vibrations are not evident in their curves when the tip of the rod was in the right auricle, the right or left ventricle. No vibrations synchronous with the first sound were picked up when the tip was pressed firmly against the ventricular wall from the inside. The intensity of the first sounds and vascular clicks recorded at the aortic orifice were less than one-fifth that

of the second sound; the first sound at the tricuspid orifice was even louder, and at this point there was no trace of click or second sound. These records *are striking proof that the first sound originates solely in the fibers of the A-V valves and those of the roots of the great vessels, and that it dies out quickly in the blood stream, in the cavities of the heart, or in the ventricular walls.*

Auscultation of the heart usually stops at the edges of the thoracic cage, although venous hums on *the neck*

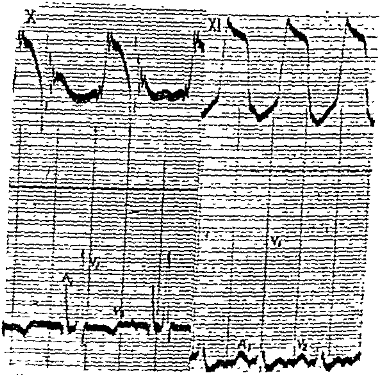


FIG 17 From MacLeod and Cohn
The left frame shows pressure close to the aortic valve, the right one, pressure in the left ventricle Audible vibrations, of low amplitude, occur with rise of pressure in the ventricle and again with rise in the aorta. The loud vibrations occur with closure of semilunar valves. None of these oscillations are recorded in the ventricle

and carotid murmurs are often sought today, and were given more attention in the nineeenth century. Josué and Godlewski pointed out in 1913 that when one listens over the right jugular bulb in a supine subject, one normally hears three sounds. The last and loudest is the aortic second sound, and the earliest and faintest is the auricular or first element of the first sound; the second sound over the jugular is the third or vascular element of the first sound. When one listens in a noisy place, only the loudest of these sounds is heard, but in absolute quiet the triple rhythm is regularly audible over the jugular. As Josué and Godlewski realized, this is a valuable auscultatory proof of sinus rhythm. In complete heart block the atrial sound is heard in varying relations to the first and second sound; in atrial fibrillation only two sounds are heard What makes all this possible is the fact that the second element of the first sound is very faint in the neck and the interval between the first and third elements is long enough to make them quite distinct. The phonocardiogram shows, however, that the atrial sound is far louder over the jugular bulb than anywhere over the precordium. In normal people it varies in intensity with respiration, tends to fade out when respiration is held, and is rarely quite as loud as the other two sounds over the jugular bulbs It often is audible on the left jugular and sometimes over the trachea. Recent studies in our laboratory have shown that this sound probably arises from stretching the walls of the veins, just as the click arises in the arteries, when there is a sharp rise in pressure This atrial sound is very loud in all cases with high venous pressure, and in such patients it varies little with respiration

The sounds which occur in diastole were originally regarded as invariably pathologic, and since they produce a triple rhythm, like the line "I galloped, Dirck galloped, we galloped all three," the term "sound of gallop" was applied to this pathologic phenomenon by Bouillaud in 1847. Heard at the apex, there are three types of gallop rhythm. In one the loudest sound is the first sound, and the two weaker sounds are the second sound and a protodiastolic sound about 0.15 sec. later. To a horseman this sounds like a horse cantering; it also sounds like dactylic verse. In another form, apical gallop consists of a faint presystolic sound analogous to the first element of the first sound, then a faint first sound, and a fairly loud second sound. Finally there may be a very loud protodiastolic gallop, with relatively faint first and second sounds. Over the jugulars of patients with high venous pressure and atrial contractions one may also observe loud presystolic sounds with fainter first and second sounds.

None of the forms of gallop rhythm sound like a horse galloping down a race track. The reason for this misnomer is no reflection on the early students of this phenomenon. In French, there is no word for "canter"; this gait is referred to as "little gallop" or *petit galop*. The English students who took over the term apparently were neither linguists nor horsemen. Fortunately, the usual sound tracks for fast action in the Western movies resemble gallop rhythm of the heart, and the cardiac gallop sounds are familiar to most medical students on that account.

The gallop sounds were heard and described by Laënnec. He ascribed the first sound to ventricular systole, the

second sound to auricular systole. In patients with ventricular hypertrophy and no abnormality in the pulse, he said that he sometimes noted two pulsations of the auricles for one of the ventricle, which is one way to acount for triple rhythm and normal pulse. Most of the early authors noted that a palpable double impulse at the apex accompanied gallop, and it was not remarkable that they concluded the sound might be due to the ventricle striking the chest wall This was Harvey's explanation for the first sound, and as recently as 1942, this theory was still being offered to account for gallop sounds. Potain, and many others down to this decade, felt that the gallop sound was due to stretching the fibers of the left ventricle as blood filled the chamber. In 1954, Dunn, of Omaha, favored a stretched pericardium as the source of the gallop sounds.

The mechanism by which the heart, in diastole and with no contraction of any muscle, can produce a sound resembling in quality and equalling in intensity the normal first sound was first described by Hirschfelder, of Johns Hopkins, and by Gibson, of Oxford, in 1907. But the phenomenon which underlies this mechanism, as both authors emphasized, was demonstrated two years earlier by Yandel Henderson, of Yale. Using Edison's recently invented cinematic camera and a heart through which saline flowed in jets, Henderson showed that each wave of rapid inflow is followed by reflux which carries the leaflets of the A-V valves back to the position of closure. This fact has been confirmed by Dean and Wiggers, using a different technique to record motion of a mitral leaflet; in the last decade the cinematic method has been

used repeatedly, and reflux closure of the mitral valve after rapid inflow has been repeatedly observed.

Hirschfelder and Gibson chose the valvular origin of the diastolic sounds because they were recording jugular pulses in healthy young people with third heart sounds, and observed a sharp rise in pressure in early diastole, following the rapid fall which accompanies outflow from atria to ventricles. The sharpness of this rise in jugular pressure paralleled the loudness of the protodiastolic sound Others had noted that the early apical thrust ended abruptly with a gallop or third sound, and that the apex then moved away from the chest wall. With these proofs that the contents of the ventricles flow away from the chest wall, and contents of the atria flow back into the veins, at the very same instant that the sound occurs, it seemed impossible to escape the conclusion that the valvular motion, described by Henderson, at times was forceful enough to tense the valve fibers so sharply as to evoke a sound. Ever since this early study, a small minority of English and American observers have accepted the valvular origin of the gallop sounds, and of sounds occurring in normal subjects at the same phase of the cardiac cycle.

Continental European and South American students of these sounds almost all prefer Potain's explanation—tensing of ventricular fibers by rapid inflow which actually stretches the muscle to a critical degree. These authors ascribe the apical retraction and rise in auricular pressure, synchronous with the sound, to the abrupt end of ventricular filling when this "limit of stretch" is reached. This explanation fits the observed facts, if two

assumptions are made. One is that there is such a limit, where ventricular filling is abruptly stopped by reaching a sharp inflexion on a pressure-volume curve; the other is that audible vibrations occur when the ventricular wall (or in Dunn's theory, the normal pericardium) is stretched to this point of inflexion.

No one seems to have bothered about this latter point Ever since hearts were first studied under the fluoroscope, it could have been shown that there was no factual basis for the assumption that third sounds and gallop sounds were due to distention of the heart to the maximum possible size. During rebound following the Valsalva experiment, normal people and patients with heart failure show large increases in heart volume, proving conclusively that no "limit of distensibility" had been reached with the smaller volume at which gallop or third sounds were noted. As shown by recent studies on dogs, and studies in our laboratory on youths with loud third sounds, the equalization of pressure between the ventricular chamber and its venous reservoir is what terminates ventricular filling. This can occur abruptly at very different ventricular volumes in different heart cycles of a dog or a man. Constrictive pericarditis alone meets the conditions assumed to underlie gallop by those who postulate a muscular or pericardial origin of these sounds.

The second assumption, that the ventricular wall, or a pericardium firmly bound to that wall, can be set into audible vibration by sudden stretching, we have recently subjected to test, and found that this assumption also lacks any basis in fact. This will come as no surprise to any student of musical instruments or of acoustic phe-

nomena. A slab of steak certainly doesn't look like a source of sharp clear sounds, even when it is bonded to a thin leather membrane, like the scarred adherent pericardium. The method of testing the sonic potential of various cardiac structures is so simple that it could have been put to the test any time since Laennec gave us his stethoscope, and objective records could have been made at any time since Einthoven made his first phonocardiograms in 1906. That the experiment was made only under the stimulus of the invitation to give the Porter Lectures, shows how warily all men avoid critical experiments, and how long they cite inconclusive circumstantial evidence.

The tests are made as follows: The base of one of the heart valves or a strip of ventricle or pericardium is firmly fastened to a piece of plywood which is weighted with lead. The other end is fastened to a similar piece of plywood with a convenient handle attached. When dropped into a laboratory sink, full of water, the weighted board sinks, the handle floats, and the strip of tissue can easily be put to stretch by sharply raising and lowering the handle. In fastening the strips, thumb tacks are used, and the edges of any strip are fastened under tension of the strip at right angles to the direction of experimental stretch. *This is so orientated as to simulate conditions in life.* Thus, in tests on atrio-ventricular valves, the annulus is fastened to the weighted board and the tips of papillary muscle to the handle. An air-tight diaphragm, held under water by a lead weight, can be used for auscultation, or connected by a large-bore tube to the microphone of a phonocardiograph (Fig. 18).

With such a simple device it is easy to obtain from the anterior mitral leaflet "first sounds" far more intense than those heard over the precordium. From the anterior (infundibular) leaflet of the tricuspid valves of human hearts, a shorter, fainter sound is obtained, while the other atrio-ventricular leaflets are much less effective sources of audible vibrations. The anterior mitral leaflet, when fastened at its free margin, instead of at the papillary muscles, gives a very short but fairly intense sound.

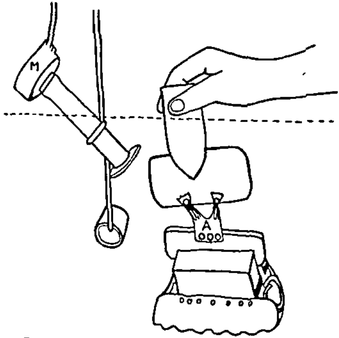


FIG 18. A gadget for eliciting heart sounds under water. [illegible]

The semilunar cusps are difficult to test because of their shape and small size.

When a strip of fat-free pericardium, 3 to 5 cm. wide and 7 to 10 cm. long, is tensed it gives off a sound almost like that of the anterior mitral leaflet with its chordae tendineae. When the strip is 10 cm. wide, has the full length of pericardial circumference from its reflections

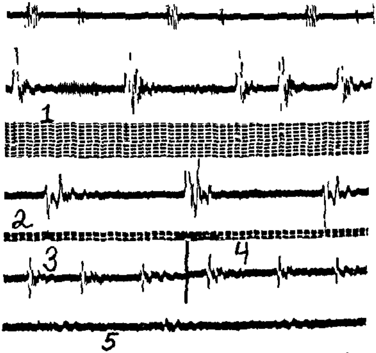


FIG. 19 Top, the apical heart sound of a normal man with the micro-
[illegible]

surface of the water

on the cavae and atria, and contains the usual amounts of fat, vessels, and nerves, the loudest sound obtained by violent tensing is dull and much fainter than those readily evoked from any atrio-ventricular leaflet. From full thickness of left ventricle and right ventricle, up to 5 cm. wide and 8 cm long, no audible sound is elicited, and no phonocardiograms can be obtained (Fig. 19). When muscle and pericardium are fastened together, so that the pericardium alone is tensed, its sound is greatly damped. We have had no opportunity to test ventricular wall with adherent pericardium. Tensing strips of the dome of the diaphragm, or of the interatrial septum, produces sounds less intense than those from the anterior leaflets, attached at annulus and free margin.

Two precautions must be mentioned. The laboratory sink filled with water transmits to the microphone any slammed door, dropped instrument, or other sharp shock in the nearby rooms. It picks up the steady hum of electric motor or running water. Late hours and holidays are best for good runs. If the handle is pulled in groups of three or four stretches, most adventitious sounds are readily recognized as being out of beat. One must use a diaphragm between water and microphone (which should never be submerged!) in order to filter out the mechanical waves set up by motion in the tank, and set the gain on the microphone to give a relatively flat base-line. We have our diaphragm three or four inches under water, and connect it to the microphone by a wide rubber tube five inches long. The records in Fig. 19 were made with no diaphragm, the tube having its lower end submerged as usual. Thus, the low-frequency waves from the ventricu-

lar strip, or from ripples due to motion of the handle, could be detected. When a diaphragm is used, no oscillations are recorded from muscle, but the vibrations due to valves are about four times the amplitude of those in Fig. 19, the same electrical amplification and microphone being used.

This experiment, easily performed with tissues obtained at autopsy, could have settled long ago the role of ventricular muscle in the genesis of heart sounds. While studies of mitral stenosis, with methods only recently available, prove beyond question the *valvular origin of the first sound and the vascular origin of early systolic clicks*, there remained some small ground for debate over the valvular origin of the diastolic sounds as long as the possibility of a muscular or pericardial origin was not eliminated by this simple test.

Most gallop and third sounds seem to be due to tensing of the A-V valves during reflux after rapid filling. The gallop in some cases of mitral insufficiency with large left ventricular and atrial volumes may actually be due to stretching of the shortened chordae tendinae when the jet of blood against the apical part of the heart causes maximal elongation of the chamber, as in B, Fig. 20. Apical retraction will occur at this instant, but inflow may continue as the ventricle "rounds out." This is the only condition in which it is possible that the A-V valve and chordae are tensed toward the end of the inflow phase and before reflux begins. Whether such a phenomenon actually exists, or whether all gallop sounds occur as a result of reflux, remains to be determined.

In myocardial failure, gallop usually is presystolic until atrial fibrillation sets in. In mitral insufficiency and constructive pericarditis it almost always is protodiastolic. Sometimes both gallops are present, or if the diastoles are shortened by tachycardia, summation gallop occurs because early inflow falls during atrial systole. In all cases, with normal third sound or gallop, the noise occurs at the end of a period of rapid flow into the ventricle. In all of these, the outward thrust at the apex is sharper than when a sound is absent and is arrested at the instant

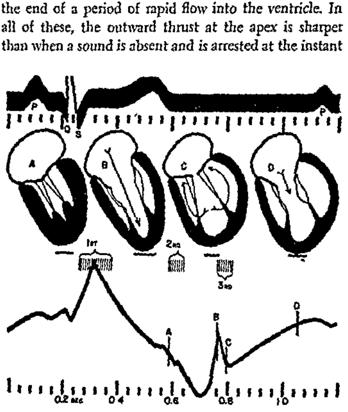


FIG. 20 From Lewis and Dock
The motion of the apex and of the heart valves and walls in relation to third heart sound or protodiastolic gallop.

the sound begins (Fig. 20). Auricular fall in pressure is reversed at the same instant. The ballistocardiogram may show sharp leftward, footward, and ventralward motion up to the sound, and a larger swing in the opposite sense thereafter (Figs. 21 and 22). These ballistic waves are larger in gallop than with third sounds in young normal persons. In many cases of gallop, the rightward ballistic thrust in diastole is greater than the systolic thrust in any plane, leaving no doubt that ample force is available, and is acting most powerfully in the direction from the ventricles toward the auricles, not in the reverse direction as

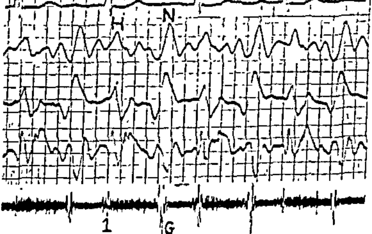


FIG. 21 From a middle aged woman with a huge heart, healed rheumatic

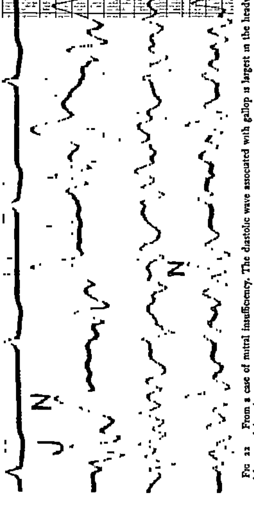


FIG 22 From a case of mitral insufficiency. The diastolic wave associated with gallop is largest in the headward and frontward directions.

would be the case if pressure toward the ventricular wall or pericardium alone caused the sound. In constrictive pericarditis and cor pulmonale, sharp jugular waves follow gallop sounds, and in those with normal third sounds these jugular waves, first emphasized by Hirschfelder, are usually present. In left ventricular failure they are often absent in the jugulars, but with the electrokymograph or esophageal pulse recorded, a rise in left auricular pressure can be shown to begin with the sound.

These sounds all occur either 0.14-0.20 second *after* the second sound, or a similar time after the onset of the *P* wave. Esophageal sound tracings often show a faint, sharp sound only 0.02-0.04 second after the onset of *P*. These tracings show a sound, or more often a split sound, 0.12-0.16 second after *P* in many cases where none is recorded from the precordium. In heart block, these sounds may be audible in the middle of the ventricular systole, so that they certainly are not equivalents of gallop Whether they arise in great veins, during rapid rise in venous pressure due to auricular systole, or come from the A-V septum as pressure differences are reversed during auricular activation and relaxation, is undetermined. The fourth or normal presystolic sound which is so often demonstrable over the jugular bulb when not found over the precordium, does seem to arise in the vein itself.

A double impulse at the apex often accompanies gallop, while in young men with third sounds and apex tracings showing early diastolic thrust and retraction, the phenomenon is rarely palpable. Bundle branch block, which may or may not cause split first or second sounds, often is accompanied for years by presystolic gallop, with

no other evidence of myocardial insufficiency, but with a striking double impulse. In the protodiastolic gallop of pericardial constriction or mitral insufficiency there may be very little systolic thrust, and the diastolic thrust and gallop may be so marked as to masquerade as systolic thrust and loud first sound. The opening snap of mitral stenosis coincides with the onset of a slow apical thrust, and is usually sharper than a gallop sound. It is heard best closer to the sternum. This sound occurs when the part of valve between annulus and adherent line of closure is drawn taut after moving toward the ventricle in early diastole (Fig. 23). When auricular pressure is high, it occurs as early as 0.03 second after the aortic second sound; when the disease is least severe it is present only after exercise and as late as 0.14 second. Catheter studies at operation show that this sound coincides with reversal of pressure between auricle and ventricle in early diastole. Since this happens earlier when auricular pressure is high, the sound and its relation to the second sound

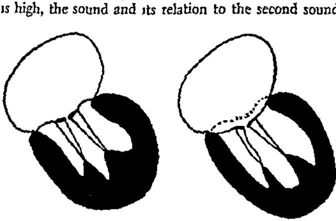


FIG. 23 From Lewis and Dock.
The motion of the atrio-ventricular septum causing opening mitral snap

are invaluable indices of stenosis and of the height of left auricular pressure due to that lesion. Catheter data from terminal pulmonary artery segements can only show how high left auricular pressure may be; it is not possible from such data to distinguish mitral stenosis from left ventricular failure. The opening snap leaves no room for doubt about stenosis; a gallop leaves no doubt that left ventricular failure is impending or present. Fortunately third sounds, unlike gallop, vary markedly with respiration, and occur only in young people with normal hearts.

The protodiastolic gallop in cases of constrictive pericarditis, and in cases of subendocardial fibrosis with moderate dilatation of the heart, may occur earlier than in cases of myocardial failure. In the latter, the volume of blood in the ventricle at the end of the systole is very much greater than in normal hearts and offers resistance to inflow. Even when venous pressure is high, inflow in myocardial failure rarely ends less than 0.12 second after the second sound When the ventricle empties more completely and the fibrous tissue in the endocardium or epicardium prevents diastolic expansion, inflow begins and ends much more swiftly than in cases of myocardial failure with equally high venous pressure. Perhaps the elastic and fibrous tissue in the wall actually hastens reexpansion of the ventricle by springing back into place like a compressed rubber bulb. In any event, the fall in right auricular pressure often ends and the gallop sound occurs as soon as 0.10 second after the second sound in cases of constrictive pericarditis with high venous pressure. Cases have been described with this interval as short as 0.08 second.

Thus, the time of occurrence of gallop and of opening snap overlaps precisely in those cases of heart failure where ventricular volume is relatively small—uncomplicated mitral stenosis and fibrosis within or about the myocardium. In the latter group, the first sound is rarely loud, but may be slightly delayed, and kymograms show very swift and early filling. Even when no mitral murmur is recorded, the slow filling of the left ventricle, apparent in apex or kymographic curves, sharply distinguishes mitral stenosis from fibrous constriction causing very early diastolic sounds.

There is one feature of cardiac auscultation more remarkable perhaps than any of the sounds mentioned. This is the systolic silence. With changes in shape and position of the auricles and ventricles, with muscle bands sliding on each other as the ventricle contracts, and with blood coursing at high velocity around the curves at the pulmonic bifurcation and the arch of the aorta, not a sound comes from the entire precordium. This leads one to believe that murmurs arise only when valve leaflets or the chordae are set into vibration, and rarely from eddy currents in the blood stream. Diastolic murmurs simulating those of mitral stenosis are often striking in patients with interatrial defects. These probably are due to vibrations set up in the edges of the septal defect when flow is most rapid. Since we wish to emphasize sounds, rather than murmurs, we will say no more about this problem.

Auscultation of the heart had little practical value as long as doctors treated all types of heart failure in the same way and had no effective therapy for bacterial endocarditis. Today, with surgical technics and antibiotics, pre-

cise diagnosis is essential, and to attain this objective with minimal lost motion, auscultation by men trained in phonocardiography is the first and most important step. An understanding of the mechanism and significance of the heart sounds is far superior to simply learning by experience that familiar precordial tunes fit certain clinical patterns and anatomical findings. The permanent objective records supplied by phonocardiograms are as valuable in managing heart disease as blood smears in leukemia, chest films in pulmonary tuberculosis, or electrocardiograms in coronary disease. Perhaps their greatest value is in teaching us to be better doctors at the bedside and in the office, and by making us less prone to error when the phonocardiograph is not available. The history of cardiac auscultation teaches us the same lesson about the mistakes made by wise and observant scientists that we are taught in every field of the history of science. Perhaps this is best summed up by Santayana:

"The wisest man contains a fool and holds a lunatic in check."

Coronary Disease — the Professor's Friend

The sprightly professor of medicine at Johns Hopkins, William Osler, wrote in his *Principles and Practice of Medicine*: "Pneumonia may well be called the friend of the aged. Taken off by it in acute, short, not often painful illness, the old escape 'those cold gradations of decay' that make the last stage of all so distressing." Twenty-one years later the aged Osler still accepted this point of view, expressed two thousand years earlier by Ecclesiasticus: "O Death, kind is thy doom to a man broken in age" With his usual whimsical jokes, the Regius Professor effectively discouraged the efforts of his colleagues to snatch him from the arms of the "old man's friend."*

In the last twenty years, pneumonia has completely lost its supremacy as the greatest cause of death of those who survive infancy; the old man has lost his most dependable friend. And though teachers and workmen, who could stay on the job regardless of old age in Osler's era, are now retired at 65 or even younger, few people have accepted that deeply religious physician's grateful attitude toward diseases like pneumonia which kill mercifully and prefer elderly victims The world was deeply shocked by Osler's last lecture at Hopkins, where he pointed out "the uselessness of men above sixty years of age, and the incalculable benefit it would be in com-

*It is interesting to note that it is only in the third edition, 1898, that William Osler recognized pneumonia as a friend In the second edition, 1895, "Pneumonia is the enemy of the aged."

mercial, political and in professional life, if, as a matter of course, men stopped work at this age."

In 1898, cancer, the slow remorseless killer, was far behind pneumonia, typhoid fever, and tuberculosis in the number of its victims. Now this cruel executioner is in second place, but the chief dispatcher of men over 40 years of age, and a common cause of death in women over 60, is coronary arteriosclerosis. This disease carries many away even more swiftly and mercifully than pneumonia. To others release comes after a short illness, much like pneumonia; while for a few men the symptoms which herald the end occur for months or even years in the brief painful seizures known as angina pectoris. Coronary disease was well known to Osler, who gave it first place as a cause of heart failure, as well as the usual cause of angina.

Osler wrote of angina, "It is a rare disease in hospitals; *it is not an affliction of the working classes. A case a year* is about the average in a large metropolitan hospital. It is a disease of the better classes [Osler meant more prosperous, not more virtuous!], and a consultant may see a dozen or more cases a year. Businessmen eating, drinking and *smoking to excess form the large contingent of angina* cases." Osler also noted the familial occurrence of coronary disease, and its many victims among physicians and teachers.

In North America there have been two great changes in the *incidence of coronary disease since 1900*. It has become far more frequent in all segments of our population and is a very common disorder in city hospitals. This *change must be ascribed* chiefly to the fact that since 1920 a very large part of the population has had a scale

of living which in Osler's day was limited to the 5 or 10 per cent whom he would have included in his "better classes." Meanwhile, the disappearance of the commonly fatal infections permitted more men and women to reach the age when coronary disease becomes prevalent.

But there has been another change as well. The age at death of males in families where this disease afflicted a father or uncle seems to have dropped about 10 years. Cases occur in so many men under 40 that it is no longer thought of as a disorder of senescence. Recent studies show conclusively that in the younger group, at any age period, coronary attacks are far more frequent in pack-a-day cigarette smokers than in non-smokers. Even among men over the age of 50, the death rate, which in either group doubles between 50 and 60, is twice as high in cigarette smokers, at any year of age, as in non-smokers. Thus, the enormous rise in consumption of cigarettes explains in part why coronary disease kills men at an earlier age in 1955 than it did before 1900.

Although the death rate has risen, the chance of dying in an attack of illness which expert physicians recognize as due to coronary arteriosclerosis has fallen in even more startling fashion. Up to 1920, the death rate in acute illness so diagnosed was over 90 per cent, and if a man recovered completely there was a strong suspicion that the diagnosis was wrong. Today the death rate in all acute episodes, when the diagnosis can be proved, is less than 30 per cent, even when we include all those who die suddenly and unattended. The chance of complete recovery, in those who survive the first week of an acute coronary attack, is about 80 per cent. Unfortunately, this change

in apparent mortality in acute attacks is due almost entirely to a change in diagnostic criteria, not to great advances in therapy such as those which lowered the mortality of various infections almost to nil. Of the 80 per cent of men with acute coronary attacks who recover completely today, probably more than 75 per cent would have recovered in Osler's day, undiagnosed and untreated.

Prior to the introduction of portable and relatively cheap electrocardiographs, most bouts of illness due to coronary disease were called "acute indigestion." In these the death rate was so low that heart disease as the cause was rarely suspected by the most astute clinicians, in the few cases which happened to be seen by such men. Another group, more seriously ill, were regarded as "acute abdomens," that is, an inflammation of the gall bladder or other upper abdominal organ, or as "pleuropneumonia" or "acute pericarditis." In these, too, the death rate and autopsy rate were so low that before 1920 the diagnosis of coronary disease was made only in those dying with a history of angina. Actually, angina does not precede most attacks. In older patients, chronic heart failure precedes death from coronary disease more often than chronic anginal seizures.

It was electrocardiography, making accurate diagnosis possible in most cases, which sharpened the ability of doctors to make the diagnosis without the electrocardiograph. Thus physicians, and even laymen, came to recognize coronary disease in all its many disguises. Thus we learned that recovery, not death or disability, usually occurred, regardless of treatment, in the many cases formerly given other diagnoses. More accurate diagnosis, controlled

by objective evidence obtained during life, is the chief factor in increasing the number of known cases. As a result of all these factors, the public knows coronary disease today as well as they knew cholera and consumption in the nineteenth century, malarial ague in the eighteenth, or plague in the seventeenth century.

The history of coronary disease is of great interest to anyone who wants to study how the human mind gropes its way through the maze of natural phenomena. William Harvey, the demonstrator of the circulation, related a typical case history of this disease, ending with rupture of the heart. Other cases were described by the great Italian, French, and English physician-anatomists of the eighteenth century. Jenner, the discoverer of vaccination against smallpox, recognized the relationship between coronary arteriosclerosis and the periodic chest pain which his colleague Heberden called angina pectoris. By the dawn of the nineteenth century, coronary disease was being diagnosed during life and confirmed at autopsy. Excellent studies of this disease were published by French and German doctors at the close of the last century.

After 1900, the great discoveries in diagnosis and in experimental production of coronary disease were reported by physicians from Russia, the United States, and Japan, showing once more that scientific progress and military power usually go together. Almost simultaneously, Obrastzow and Straschesko in Russia and James Herrick, of Chicago, described the brief illness, with chest pain, fever, leucocytosis, and features resembling pneumonia or acute abdominal inflammation, which has become known as "acute coronary occlusion with myocardial

infarction." Both these reports emphasized the possibility of complete recovery, and noted that healed cardiac infarcts were not infrequently found at autopsy of men who died of other causes. Neither report had much influence on medical practice or on textbooks, for the only cases proved to have such attacks were those who died, and doctors all thought of coronary disease as a fatal illness.

In 1918, Herrick reported electrocardiographic changes occurring in myocardial infarction, and his associate, Fred Smith, showed that ligation of the coronary arteries of dogs caused similar changes. Within three years, Frank Wilson, in Michigan, Samuel Levine, in Boston, and H. E. B. Pardee, in New York, had fully confirmed and expanded the electrocardiographic features of coronary disease. Since then the changes have had the same relation to this disease as the Widal test in typhoid, or the Wassermann in syphilis. Only recently La Due has reported the rise of glutamic transaminase in the blood plasma which occurs with necrosis of heart muscle, and thus given us another method of proving that acute myocardial infarction has recently occurred.

Those who used the electrocardiograph soon found that recovery, not death, was the usual outcome, and that infarction with minimal or no symptoms could be detected. Thus they became skilled in making the clinical diagnosis before the confirmatory traces were available, and even in those who had no electrocardiographic evidence The latter group has fallen from 20 per cent with the original limb leads, to about 8 per cent with the conventional 12 leads in use for the last 15 years. This knowledge only gradually made its way into professional con-

sciousness When President Harding had a typical coronary attack, in 1923, he was considered by San Francisco specialists to be suffering from gall-bladder disease and pneumonia. When Sir Thomas Lewis, the greatest English authority on electrocardiography, had a typical attack in 1926, he and his colleagues first diagnosed acute indigestion, later acute pericarditis

By 1930, the knowledge of this clinical picture, and of its electrocardiographic confirmation, had become worldwide, as had the sale of portable electrocardiographs. Overdiagnosis became common, as trivial changes in the electric trace were misinterpreted. Cases of pulmonary embolism, post-infectious myocarditis, or those with no organic disease, were put to bed for the six weeks which had become the conventional treatment for a disorder from which 80 per cent of the patients would recover with no treatment at all. Today, the reaction has set in, diagnoses are more cautiously made, and treatment is fitted to the patient and his clinical findings, of which the electrocardiograms are only a part

One does not know whether to be more astonished by the speed with which experimental pathologists established the pathogenesis of coronary disease, or the obstinacy of the practicing physician and cardiac specialist in ignoring or denigrating this work. It is the familiar story of puerperal sepsis all over again—the cause and prevention of a disease clearly proved and the experts in the field ridiculing the whole thing and insisting that puerperal sepsis, tuberculosis, or coronary disease are not preventable but mysterious manifestations of God's will, and beyond all human control

During the nineteenth century, pathologists observed that the patchy yellow thickenings which appear in the linings of human arteries contained fatty material, as did injured livers, anemic heart muscle, damaged kidneys, or parts of the brain affected by strokes. So did the older masses of dead tissue in the large tuberculous lesions. It was concluded that all these were signs of tissue degeneration, a sort of spontaneous decay of the body. The great pathologist Virchow, in his old age, was as skeptical of the contagious nature of tuberculosis as the obstetricians of Budapest and Philadelphia had been of Semmelweis' or Oliver Wendell Holmes' idea that childbed fever was contagious.

It was the high incidence of puerperal sepsis in teaching wards, and the low incidence in women delivered at home, which led acute observers to look for further proof of contagion. Finally, Pasteur and others described the hemolytic streptococci which caused the fulminant type of childbed fever. It was the low incidence of arteriosclerosis among the poor and those who lived largely on vegetables and grain, the high incidence among the rich and especially among pastoral people who lived largely on whole milk, that led to the suspicion that diets rich in animal products cause atherosclerosis. This theory was tested in 1908 in Russia, where Ignatowski reported production of the disease by feeding rabbits diets of meat, milk, and eggs. Whether it was the protein or fat remained to be tested, but by 1910 it was shown that the protein fraction was not effective. By this time, French and German chemists had begun to study the cholesterol in plasma and tissue, and Windaus discovered that arterio-

sclerotic plaques were rich in this animal wax. Very promptly Anitschow in Russia and other workers in Germany reported that feeding rabbits cholesterol caused the arterial disease. Only five years elapsed between testing the effect of animal products and proving that cholesterol in the diet probably was the cause of coronary disease. The well-known social and geographic variations in incidence of arteriosclerosis were explained by showing that cholesterol, abundant in the diets of those with severe disease, absent or scanty in the diets of those free of lesions even in old age, could cause the disease in a susceptible animal species

All of the arguments raised against Villemin, when he reported transmission of tuberculosis to some rabbits, were soon raised against Anitschow. The lesions and course of the disease were different in the animals and in man, the dose used to cause the experimental disease was beyond anything man experiences, and in most laboratory animals the experiment fails. Only guinea pigs were suitable for experiments on tuberculosis or scurvy, only rabbits for atherosclerosis Therefore, the animal experiments have no meaning for physicians.

In 1922 Yamaguchi reported producing arteriosclerosis in chickens by feeding them egg yolk, and Dauber, Stammler, and Katz have repeated this, using young chicks and pure cholesterol supplements. Others have produced lesions by feeding cholesterol to dogs (but only on diets poor in protein or after reducing thyroid function) and in the Cebus monkeys (but only on diets poor in methionine) Though arteriosclerosis in animals resembles that in man more than tuberculosis in guinea pigs

resembles human pulmonary tuberculosis, many great authorities still *reject the animal experiments as evidence* for the importance of dietary cholesterol in arteriosclerosis.

Although arteriosclerosis has not been produced in any mammal on a low cholesterol diet, two groups of leading clinical authorities, each with an outstanding expert on experimental atherosclerosis, agreed in 1954 that dietary cholesterol was of no importance in the human disorder! By then it had been shown that the experimental disease was greatly accelerated by high-blood pressure, in dogs, rabbits and chickens, just as is the disease in man. Lober, in Minnesota, had shown that deposition of cholesterol in the coronaries is progressive throughout life, and is unusually rapid in the first year of infancy, while Bragdon had reported that newborn rabbits have the same rise in plasma cholesterol in the first days of life as do human babies, and that suckling rabbits, like our infants, have progressive deposition of cholesterol in their arteries, which disappears after weaning. Rabbit milk is three times richer in cholesterol than human milk. It was conceded that atheromas are formed by cholesterol *from the plasma, and that plasma cholesterol, and* especially the fraction bound to beta globulin, is especially high in those men who developed coronary diseases before 50, and in the animals and birds which develop the disease on cholesterol feeding. Yet none of these facts impressed the learned panelists of the New York Academy of Medicine or the panel of the College of American Physicians. Most of these authorities thought that obesity, per se, might be a cause of arteriosclerosis. This in spite of the

fact that intensive studies in the Army, in Westchester County, and in Boston had shown no significant difference in the percentage of coronary victims, and victims of accidental trauma, who were of ideal weight, underweight, or overweight. Thus the panelists preferred a mysterious theory of pathogenesis for which there was no clinical or experimental evidence, and rejected one for which there was suggestive clinical and an enormous experimental support.

In World War II, deaths from coronary disease in the American armed forces, in men under the age of 35, appear to have been nearly ten times as frequent, per man-year, as in the British forces, and probably were more than a hundred times as frequent as in the Japanese services. Studies on the coronary arteries of soldiers dying of wounds in Korea showed that while Koreans had no lipid infiltrations of significance and no encroachment of intimal cushions on the lumina, 10 per cent of Americans, with an average of 23 years, had the lumen of one major coronary reduced over 50 per cent by plaques rich in cholesterol. At these points, the reduction of luminal cross section was 80 per cent, on the average. The extraordinary differences in incidence of coronary atheromas in soldiers of various armies have been ascribed to psychologic factors, to constitutional differences, but rarely to the great differences in the army diets.

It is true that we can produce severe and permanent [illegible]

[illegible]

the same age. The difference in blood pressure levels [illegible]

Japanese, British, and American fighting men certainly does not account for the difference in coronary disease, and other ways in which the psyche can damage the arteries remain unproved.

There certainly are great differences in constitution and metabolic patterns between the white race and the races which originally occupied the New World and northeastern Asia. But when American Indians or Asiatics are put on the diets which are usually given to American troops, they show the same levels of plasma cholesterol as do soldiers of the white race. While constitution may alter the tendency of arterial disease to localize in coronary vessels or of diets to alter cholesterol patterns in the blood and cholesterol deposition in the arteries, there is no proof that this factor is of importance in accounting for the differences in incidence of coronary disease in soldiers of various armies.

The Japanese diet, even for men in the army, was extremely low in cholesterol; the British army diet has only a modest content; while the American officers and enlisted men receive diets richer in cholesterol than that of any large group of men anywhere in the world. Knowing this, and knowing that rabbits, dogs, and chickens can develop these lesions only on diets rich in cholesterol, the average layman is forced to one of two conclusions—either the disease in man is due largely to diet, or animal experiment, which is in accord with observations on man, is of no value in elucidating the cause of disease. The conclusions of the two panels certainly would rejoice antivivisectionists These physicians tacitly agreed that the innumerable rabbits, dogs, monkeys, and chickens used

in studying the pathogenesis of arteriosclerosis had died in vain, since their lesions were not relevant to man.

It is well at this time to review the reasons given by most students of coronary disease for rejecting dietary cholesterol as the fundamental factor in causing human arteriosclerosis. They all state that dietary cholesterol is unimportant because men normally synthesize this material in their tissues in quantities comparable to that in a rich diet. This, of course, was known to Anitschow and all subsequent students. Rabbits, dogs, and chicks, indeed all animals, synthesize cholesterol, and cows and hens put out large amounts daily, in milk or eggs, on cholesterol-free diets Diabetic rabbits given detergents by vein, greatly increase cholesterol synthesis and, on cholesterol-free diets, have blood levels 20 to 40 times their normal 60 mgm. per cent,* or the 200 mgm. per cent due to suckling or to adding pure cholesterol to a diet. Yet these endogenous hypercholestrolemias cause no atherosclerosis. Since endogenous cholesterol is unrelated to experimental disease, and synthesis is more active, in mgm per kg per day in chicks and rabbits than in men, there is no reason to deny the importance of dietary cholesterol in men after proving that it is the essential cause of experimental arteriosclerosis Endogenous cholesterol production is a universal phenomenon in vertebrates, but it does not lead to atheroma formation in any mammal; dietary cholesterol does.

Another, and apparently a more valid, reason for doubting that man responds to dietary cholesterol as do

* The standard clinical expression 100 mg per cent means 100 mgm. per 100 ml. or 1 mgm per ml. of blood

experimental animals, is that men on diets rich in cholesterol often have blood levels much lower than chickens, rabbits, or dogs on atherogenic regimes of cholesterol feeding. Of course, the animals develop severe lesions in weeks or months, men in decades. The blood level is more stable, with varying cholesterol intake, in man than in rabbits or chicks. But in Brooklyn, where high cholesterol intake is customary, blood levels usually are over 250 mgm. per cent, and at autopsy in the City Hospitals, *40 per cent of women over 40 years of age have cholesterol* gallstones and 50 per cent of the men have striking coronary atherosclerosis by 50 years of age. In Costa Rica, *in Peking, Hong Kong, and Okinawa, autopsies on similar* material show almost no cholesterol stones and few atheromas; there the blood cholesterols are under 160 mgm. per cent and the diets are almost cholesterol-free.

We can offer no explanation for the notion that obesity as such causes atherosclerosis. Undernourished rabbits, given cholesterol, develop higher blood levels and larger plaques than well-fed controls. The half-starved Hindus, who use butter as their chief source of fat, show far more atheromas than well-nourished or obese Okinawans, who use bean oil Obesity due to low cholesterol diet has never produced either experimental or human atherosclerosis.

Some very bizarre human feeding experiments are cited to show that it is the fat content, not the cholesterol, which influences human plasma levels of cholesterol and thus alters rates of development of coronary disease. Plasma cholesterol falls sharply when [illegible] placed on low-fat, low-protein cholesterol-[illegible] as consisting of rice, [illegible] sugar. [illegible]

free of cholesterol, is added, the plasma cholesterol rises again. Results of this experiment seem to be incompatible with the data obtained by studies on groups of food faddists, who live either on purely vegetable diets, or on vegetables plus eggs and milk. The real vegetarians have low blood cholesterol, even though 30 per cent or more of their calories are derived from fat.

It is known that bile stasis and low rates of excretion of biliary cholesterol result from diets low in fat. Reabsorption of biliary cholesterol is high when the diet contains reasonable amounts of fat, and this cholesterol presumably acts in the body in the same way as dietary cholesterol. Thus, adding fat to rice diets actually raises cholesterol absorption Rice diets are very low in protein, and the experiments of Li and Freeman showed that on protein-deficient diets, cholesterol added to the food caused marked rises in plasma levels It seems that the paradoxical result of adding fat to rice diets is due to the slowing of metabolic processes due to protein deficiency, and to high reabsorption of biliary cholesterol due to fat indigestion. These experiments do not prove that elevated blood cholesterol levels in people on the American high-fat, high-protein, high-cholesterol diet are due to the fat and not to the cholesterol. Other experiments have shown that fat, as such, does not raise, and that vegetable fats usually lower, plasma cholesterol when added to diets with average fat and protein content.

Thus, Hardinge and Stare found that plasma cholesterol was 34 per cent higher in non-vegetarians, and 30 per cent higher in vegetarians using eggs and milk, than in true vegetarians. These were all young adults, all the

diets contained 80 gm. of protein, and over 30 per cent of the calories came from fat. The cholesterol intake was 900 mgm. a day in the non-vegetarians, 330 in the ovo-lacto-vegetarians, and zero in the pure vegetarians. These and other data indicate that once dietary intake is over 300 mgm. per day, plasma cholesterol rises very little for each 100 extra mgm. in the diet.

Students of Oriental diets and pathology have suspected that vegetable products and especially vegetable oils may actually have a protective action, minimizing the effect of modest quantities of cholesterol in the diet. This has been confirmed by showing that the sterols in grain and other vegetable products, fed to rabbits and chicks, prevent atherogenesis or the rise in plasma cholesterol which usually follows cholesterol feeding.

Ahrens, at the Rockefeller Hospital, found that a group of volunteers, all obese, who were kept on diets which prevented weight loss, had a rise of 30 per cent in blood cholesterol on switching from a diet with 40 per cent of the calories derived from vegetable fat to a similar animal fat level. Adding cholesterol to the vegetable fat, to bring it up to the level of 1 to 1.4 gm. which was contained in the animal fat regime, failed to produce the rise affected by the animal fat. This clearly establishes a protective effect of vegetable oils and confirms earlier observations of reduction in blood cholesterol of men and dogs when vegetable fat was given in large quantities.

It will be interesting to see how the opponents of the dietary cholesterol theory of atherogenesis evade these new facts, but we can be sure that they will refuse to accept even crucial experiments on man, and will still in-

sist that in man dietary cholesterol is not related to blood cholesterol or to atheroma formation. They have fought the good fight for over forty years, and will not be impressed by Ahrens' observation of blood cholesterol progressively falling as the intake of corn oil of his subjects was raised to provide up to 70 per cent of total calories. Thus did the Paris obstetricians greet Pasteur's report on puerperal sepsis; thus did Virchow dismiss Koch's work on tuberculosis.

For the student of medicine it is important to understand that the slow spread of knowledge of the clinical picture of acute myocardial infarction and the deliberate rejection of dietary cholesterol as its cause are not evidences of the stupidity of particular men, but show how hard it is for all of us to accept new facts, especially if they lead to disagreeable conclusions What conclusions could be more annoying that that drawn from the experiments of Ignatowski and Anitschow, of Stare and Ahrens? Can we believe that it is not the hard work and high tension of the rich man or rich nation but a delightfully rich diet which leads to early death from coronary disease? We cannot bring ourselves to believe that diets full of eggs and milk, which nature had never intended to be used for human food, might be harmful as well as delicious. We had a hard time in coming to admit that bad diets caused scurvy, pellagra, and beriberi, and it is even harder to realize that our civilization has reached the stage which the Romans enjoyed when Lucretius wrote, "In primitive times, lack of food gave languishing bodies to

Death; now, on the other hand, it is abundance that buries them."*

As of 1955, the theory of Ignatowski seems fully confirmed—men will not develop arteriosclerosis on vegetarian diets, no matter how liberal in calories or in fat. They will develop such lesions on diets rich in eggs, butter fat, or animal fats of any kind The vegetable diets reduce, the animal fats augment, the rise in plasma cholesterol *due to absorption* of dietary or biliary cholesterol from the intestine. Diets low in protein but containing fat of any kind permit intestinal absorption of biliary cholesterol but delay its removal from the plasma.

Individuals, like animal species, vary in the way they respond to diet, so that a regime which raises one man's plasma cholesterol to 500 mgm. per cent will leave another's at 300, or even at 150. One important factor is the metabolic level due to thyroid activity; perhaps equally important is that due to physical activity. London conductors, running up and down the two-decker stairs all day to collect fares, develop coronary disease later in life than bus drivers, sitting all day at the wheel. George Mann observed a marked rise in serum cholesterol in young men when, on constant diets high in calories and animal fat, they changed from daily strenuous exercise, which prevented gain of weight, to a sedentary life with deposition of fat.

One of the most curious features of this whole problem is the fact that men under 50 have far less cholesterol in the gall bladder than do women, in countries where

* *tum penuria deinde cibi languentia leto*
membra dabat, contra nunc rerum copia mersat.
—De rerum natura, V, 1097

the diet is rich in cholesterol, while women under 60 have far less cholesterol in their coronary arteries. In the aorta, atherosclerosis develops at about the same pace in men and women, and also in chickens, which show the same sex difference in coronary sclerosis as do human beings.

In male chicks, estrogen administration retards coronary but not aortic sclerosis due to cholesterol feeding, and in men, estrogens cause the fraction of cholesterol bound to beta globulin to fall to the level usual in young women. In the chicks, estrogens raise plasma phospholipids without lowering the cholesterol content. It now appears that cholesterol can precipitate out in the skin, but not in the blood vessels, when cholesterol and phospholipid are both very high, as in chronic biliary obstruction; it can precipitate out in the aorta when cholesterol is much lower but the cholesterol-phospholipid ratio is higher, and in the coronaries only when the ratio of cholesterol to phopholipid reaches the very high levels observed in chick or man when a liberal cholesterol intake is combined with a high androgen-estrogen ratio of endocrine activity.

The predisposition of coronary arteries to atherosclerosis has always been a striking phenomenon in young men. In the American soldiers who died of coronary disease, there were often many plaques in these vessels, none elsewhere in the body. In others, a single strategically placed lesion, no larger than a grape seed, may have caused death, while many old men and women have dozens of larger, but harmless, coronary plaques and their aortas show scores of huge lesions, up to two or three inches long. Obviously chance plays a large role in de-

termining which person on a high cholesterol diet will become ill or die at any given age, even in a group all of whom are depositing the same quantity of cholesterol in their arteries each year.

Because the coronaries supply so vital an organ as the heart, a lesion here may cause serious disease when a similar one in many other arteries of the same size would have trivial effects. Actually, similar lesions in other arteries are very rare, and during the period of active estrogen production, women with gallstones and aortic plaques have relatively little coronary disease. Therefore, the predisposition of coronary arteries to atheroma formation, when estrogen levels are relatively low, is a factor almost as important as dietary cholesterol in causing the prevalence of coronary disease.

There is an obvious anatomical basis for coronary disease. In men and animals these arteries have remarkable "cushions" of intimal thickening, which unfortunately are sometimes described as "lesions," free of lipid (fatty materials, including cholesterol and compounds containing phosphorus and fatty acids), in studies of human or animal tissues. These cushions run longitudinally and contain smooth muscle fibers which also run parallel to the lumen, unlike the spiral strands in the media which run almost at right angles to the lumen. The cushions contain fine elastic fibers and loose collagenous tissue. Even in newborn babes, such a cushion may be two or three times as thick as the media. These structures occur in other arteries (occipital, penile) which frequently alter their length, as the coronaries do at each heartbeat. They are absent in most of the arteries of the body. They are more

numerous and extensive in men, or in newborn males, than in females. This fits the fact that even before birth males have slower heart rates and bigger stroke volumes than females. The percentage of change in length of these arteries with each heartbeat is larger in males than in females, so the anatomic feature peculiar to arteries of changing length is more striking. Perhaps chemically, as well as structurally, the coronary intima tends to precipitate out plasma lipids in a different way than the intima of the aorta or other arteries. Why it takes up these substances when the lipid pattern is that determined by the male steroid levels is unknown. In any event, the predisposition of men to coronary disease has at least three bases: men eat more animal fat, they normally have thicker coronary intimas, and their blood lipid pattern specifically favors coronary precipitation of cholesterol when total cholesterol level is the same as that of women.

While our knowledge of cholesterol metabolism, of plasma lipids, and of atheroma formation is only rudimentary, we probably can explain why men in North America have severe and precocious coronary disease better than we can explain why some people develop virus diseases, tuberculosis, or pneumonia, when most of those who pick up the agents causing these diseases remain well. Even now it can be said that we know how to wipe out coronary disease just as effectively as we have wiped out cholera, yellow fever, plague or typhus, and more completely than we have wiped out pneumonia, tuberculosis, or syphilis. It is even now conceivable that by adding vegetable sterols and vegetable oils to such processed foods as milk and candy we could greatly reduce the inci-

dence of gallstones and coronary disease without changing our diets.

If our knowledge of coronary disease were put to practical use, 30 per cent of us, who now die of this acute, short illness, could expect instead the slow torture of cancer, or the "cold gradations of decay" described by Osler as the alternative to pneumonia. Certainly those who believe "one crowded hour of glorious life is worth an age without a name" will be reluctant to give up the certain solace of inhaling clouds of tobacco smoke, or enjoying good food, in order to postpone a coronary death, which in any event awaits only one in three of us American physicians, and gives the non-smoker only ten extra years.

It is unfortunate that coronary disease today, like pneumonia in Osler's time, kills many men with dependent wives and children. Neither of these great killers limits its victims to old people, threatened with dismal senility. It is the great friend of sedentary men, and therefore of that most sedentary of all men, the professor. Osler chose a family of professors, in which father, son, and grandson all died of coronary disease, to illustrate the hereditary factor. But it also kills many professors before the age of 60, at which time Osler felt they had best fade away. It thus becomes a very different sort of friend of the faculty, by making possible a whole series of promotions—an associate moves into the chair; an assistant moves up a notch; an instructor gets a post with tenure; and three wives find it easier to meet their bills. Only the automobile can be compared with coronary disease as an agent which suddenly and frequently raises the standard of living of three families while casting one into gloom.

So long as coronary disease seemed a mysterious and capricious accident, or even the result of hard work and worry, it was not easily accepted as one of nature's curious blessings. But now that we know it to be due to a luxurious way of life, and to the happy hours spent with Lady Nicotine, there is little reason to complain when, in our own lives, we experience its first warnings. Even if we don't go near a doctor, the odds strongly favor our complete recovery from this brush of the angel's wing If we change our diets in a sensible way, we can expect to avoid further attacks and to continue to work and play as vigorously as ever. Even if we do not change our habits, we may go along comfortably for many years after a coronary attack. However, with the knowledge we now have, many may choose to live cautiously on the diets normal to man, rather to persist in a way of life "contrary to nature," which has brought us into peril of death. And if one of us, well aware of the facts, chooses to continue on the primrose path, and finds the Professor's Friend taking him swiftly from his chair, he will always have the consolation of knowing that his family's loss is compensated by creating opportunities and reducing economic pressure for at least three younger families.

In addition to offering this grain of comfort, and showing you the silver lining in the ominous cloud which hangs over the middle-aged or aging professor, one may point out how greatly the disability due to myocardial infarction has been reduced by the increasing acceptance of the Frank Wilson program of therapy, "confinement to quarters," rather than the original six-weeks-of-bed-rest, six-months-off-the-job program. If a patient is in shock, or

under deep narcosis for pain, he usually must be kept in bed for hours or days. But if he has milder pain or difficult breathing without shock, he can be made more comfortable by being allowed to move gently from bed to armchair to bathroom (or commode) and back to bed again. If he needs oxygen but is not in shock, his circulation may be less embarrassed on this program than if he is kept flat in bed. Usually he is comfortable in a few hours, or at most in a few days, even though he has fever. If he is confined to quarters rather than to bed, he will sleep better, have less indigestion and discomfort, and will not lose his vasomotor control and become dizzy when he finally is allowed to move about. He never will become weak, and will not require rehabilitation over several weeks in order to walk in comfort. While his return to stair-climbing and other efforts may not be permitted for four to eight weeks, he usually is well enough to carry on most of his normal activities within a few weeks and may be fully restored to all his usual pursuits within two or three months. In the milder episodes of coronary disease, capacity for sedentary work may be fully restored in days or weeks, and the whole disease, from the standpoint of patient and family, becomes far less alarming, disabling, and economically catastrophic than it usually was before the cautious pessimism of earlier therapeutic programs was replaced by a more physiological and humane method of management.

One can now assure the patient and his family, no matter how serious the initial illness, that if the patient does well through the first few days there is an excellent chance he will be comfortably convalescent in a week,

and fully restored to his normal way of life within two or three months, perhaps even sooner. As with most illnesses, our knowledge of proper care of coronary disease lagged behind our diagnostic skill, and even behind our slow acceptance of the knowledge of its cause and prevention.

For reasons which are obvious to anyone who likes American cooking, it is unlikely that our knowledge of the prevention of coronary disease will affect the incidence of this disease, even among physicians, in the next few years. Even the rational management of the acute illness is now viewed with grave suspicion by most internists and cardiologists, although it was practiced by Frank Wilson and has been urged by Samuel Levine and Tinsley Harrison for some years. The 1955 edition of a popular and authoritative textbook of medicine suggests that a patient with a "mild uncomplicated attack" may be permitted increasing periods in a chair in the "fifth and sixth weeks." Ice-cream, milk and eggs are recommended as useful in dietary management of coronary disease in its acute phase, nothing is said about dietary cholesterol in pathogenesis or management of coronary atherosclerosis. But even in this text, another contributor refers to the reports of a "predictable reduction of serum lipids with the use of diets containing vegetable fat but no animal fat." Obviously, we are just at the turning point in the history of coronary disease, when two equally popular and authoritative texts give diametrically opposite accounts of the role of diet in pathogenesis and of the value or disadvantages of prolonged complete bed rest in the management of coronary disease. Today one can

cite good authority for almost any program of managing one's prospective encounter with a coronary attack.

No matter how we manage the onset of this disorder, if we continue our usual diet we can be confident that the Professor's Friend, coronary atherosclerosis, will spare one-third of all professional men the specter of senility with its "cold gradations of decay," or cancer with its painful erosion of body and mind. At an earlier age, this popular masculine complaint will give many of us that intimation of mortality which makes life sweeter and more vivid, and an excuse to loaf, avoid committees, and be treated for a few weeks with unusual respect. Let us, then, learn how to live on the best possible terms with coronary disease, the Professor's Friend. When he arrives for a brief visit or a longer stay we can choose between being confined to our quarters with him, or keeping him as a bed-mate for weeks or months. Should he come to escort us to Charon's bark, may we be able to say with Horace, "I have lived my day; tomorrow let our Father fill the sky with black clouds or the flawless sun!"*

* laetusque degit, cui licet in diem
dixisse "Vixi: cras vel atra
nube polum pater occupato
vel sole puro, non tamen irritum,
quodcumque retro est, efficiet, neque
diffinget infectumque reddet,
quod fugiens semel hora vexit."
—*Odes*, III, xxix, 41-48

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rate was twice as high, in mild or severe attacks, if strict bed rest was enforced. The classical treatment, in other words, leads to doubling the death rate in hospitalized patients, as compared with a "confinement to quarters," without enforced bed rest.

Index